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## NINTH SYMPOSIUM NEURORADIOLOGICUM GOTHENBURG 24—29 AUGUST 1970

*In this special issue we have the privilege of publishing the majority of papers presented at the Ninth Symposium Neuroradiologicum in Gothenburg. The papers are grouped according to the subject matter and the aspects from which this is treated. Some of the contributions have therefore not been included in the same subject groups as in the Symposium programme. The classification of certain papers such as those dealing with comparisons between different methods of examination may be a matter of opinion but we have endeavoured to place each paper under the heading that seemed most appropriate. In each group the papers are arranged in alphabetical order according to the first author's name. The titles of the Symposium papers not published here are included in the Table of Contents under the sections in which they appeared in the Symposium programme.*

Biologic and therapeutic communications are published in *Acta radiol Ther Phys Biol* 11 (1972) 329

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*The Editor*

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CONVENTIONAL EXAMINATIONS OF THE SKULL  
OR SPINE



## CYTOMEGALIC INCLUSION DISEASE

### Report of a case diagnosed radiologically

by

V. GVOZDANOVIC, Lj. ZERGOLLERN, P. ERAK, S. ŠIMUNIĆ and R. CRNOJEVIĆ

Pathologic changes in cytomegalic inclusion disease were described as early as 1882 by RIBBERT although MERCER et coll. were the first 17 years ago to publish radiologic evidence of intracranial calcifications. The number of verified cases of this condition in the roentgen literature is however, still small and opinions differ about the possibility of its radiologic differentiation from other intracranial calcifications.

Cytomegalic inclusion disease is an asymptomatic virus condition of the salivary glands not uncommon in country people in contact with animals. Infection of the child occurs mostly in the fourth month of fetal life and attacks various organs. The brain is affected only in 20 per cent of cases, meningo-encephalitis develops in the matrix around the lateral ventricles the immature tissue being more sensitive to virus infection. The process is often combined with haemorrhages and followed by atrophic changes. The calcifications in the necrotic and haemorrhagic tissue may appear immediately post partum although usually in the first two to three months of life. The lateral ventricles are often symmetrically dilated as a consequence of the periventricular atrophy. Should adhesions





Fig 1 a Extensive intracranial calcifications symmetrically distributed b Structure of the calcifications in the lateral tomogram

in the posterior fossa develop dilatation of the ventricular system will be more extensive

Intracranial calcifications are radiologically evident in a small percentage of cases only. They are usually bilateral, symmetric and distributed perivascularly mostly in the subependymal periventricular tissue and the basal ganglia. Calcifications may also be present in the cortical and subcortical regions. DAURELLE *et coll.* (1958), ALLEN & RILEY (1958), HAYMAKER *et coll.* (1954), SACKETT & FORD (1956) believed that the distribution of the calcifications described is characteristic. TUCKER (1961), MOLLOY & LOWMAN (1963), TAVERAS & WOOD (1964), CAFFEY (1967), OEHME (1957), STAMPAK PLASAJ & URBANKE (1962) considered that roentgen differentiation between cytomegalic inclusion and toxoplasmosis is hardly possible.

The differential diagnosis between intracranial calcifications in generalized herpes zoster and idiopathic intracranial calcifications should be mentioned.

### Case report

Girl aged 6 months with marked psychomotor retardation. First child of healthy young parents; pregnancy and birth normal. She developed right facial paralysis when she was 2 1/2 months old and was admitted to hospital for two months with encephalitis of unknown origin.

Slight microcephaly noted. Motor reaction was poor and the child generally flabby. The muscles of the upper limbs were hypotonic and those of the lower extremities hypertonic with increased deep reflexes. Tests for psychomotor development corresponded to those of a 2-month-old child. EEG suggested severe damage to the brain. RVA herpes test 1/64. Cerebrospinal fluid normal.

Skull films demonstrated extensive intracranial calcifications in the *ap* projection (Fig. 1*a*) lying symmetrically distributed in a letter delta formation. In the lateral projection they were less obvious and localized mostly in the parietal region in a rounded area of 8 cm diameter. Lateral tomograms (Fig. 1*b*) indicated the radial and honeycombed character of the calcium deposits; some were worm-like as in Sturge-Weber disease. Encephalography with 15 ml of air disclosed moderate symmetric dilatation of both frontal horns; other parts of the ventricular system including the third and fourth ventricles were normal. The calcifications were situated periventricularly (Fig. 2). The tomograms demonstrated the subependymal periventricular distribution of the calcium deposits in the corpus callosum that formed the roof of the delta (Fig. 3).

The finding of extensive symmetric calcifications in a child of 6 months suggested cytomegalic before encephalography strengthened the tentative diagnosis. The distribution of the calcifications was unlike that in cases of toxoplasmosis previously observed. Definitive proof was obtained by *unne* analysis. The inclusion body cells — sometimes likened to the eye of an owl — persisted throughout the three-month stay of the child in hospital.



Fig 2 Ap and pa encephalograms. Moderate symmetric dilatation of both frontal horns. The calcifications lie periventricularly.



Fig 3 L encephalogram obtained during encephalography. Subependymal distribution of the calcifications.

### Conclusion

The distribution of the calcifications, their structure and the symmetric dilatation of the frontal horns in this case suggested cytomegalic inclusion disease rather than toxoplasmosis. The diagnosis of the former is made essentially in the laboratory, so that the radiologic differentiation is not of prime importance. Intracranial calcifications in a newborn or young child should however indicate tests to confirm or eliminate cytomegalic inclusion disease.

## SUMMARY

The examination of a child with signs of cytomegalic inclusion disease (a virus infection of the salivary gland of animal origin) is reported. The tomographic appearances of the intracranial calcifications are described and the differential diagnosis is discussed.

## ZUSAMMENFASSUNG

Die Untersuchung eines Kindes mit Zeichen einer cytomegalen Einschlusskrankung (eine Virusinfektion der Speicheldrüse animalen Ursprungs) wird vorgelegt. Das tomographische Erscheinungsbild der intrakraniellen Kalkifikationen wird beschrieben und die Differentialdiagnose besprochen.

## RÉSUMÉ

Les auteurs présentent l'examen d'un enfant atteint de signes de maladie des inclusions cytomegaliques: infection virale des glandes salivaires d'origine animale. Ils décrivent l'aspect tomographique des calcifications intracrâniennes et en étudient le diagnostic différentiel.

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## CRANIOSTENOSIS AND DIPLOIC LAMINATION FOLLOWING OPERATION FOR HYDROCEPHALUS

by

J W LOOP and E L FOLTZ

DE BOLLAY in 1963 called attention to occasional radiographic changes in the skull following hydrocephalus operation. His monograph illustrated lamination of the diploe, thickening of the vault and premature fusion of sutures. ANDERSSON in 1966 reported three cases of premature synostosis of after ventriculo-venous shunting and cited other examples which had come to his attention. MOSFLEY et coll (1966), LEGRANGE & SCHAFER (1966), KURLANDER & CHUA (1967) and KLOSS (1968) have noted additional cases.

Craniostenosis was not reported as a complication of the surgical treatment of hydrocephalus in 50 cases by YASHION & SEGAR (1964), in 109 cases followed by JOYNS (1967), or in 455 infants treated by ventriculo-venous shunts over a five year period by FORREST & COOPER (1968).

In our own experience radiographic abnormalities such as those described by DE BOLLAY are not infrequently seen following ventriculo-atrial shunting in infancy. We have undertaken a review of skull films and clinical records in an attempt to define the prerequisites and correlates of these changes.

*Method and Material.* Case records for 111 patients treated consecutively in our clinic with ventriculo-atrial shunting using the Pudenz Heyer apparatus were reviewed. A selection of cases for this investigation was made to include all



Fig 1 A view of the bregma and frontal bone in a patient two years after hydrocephalus operations reveals fusion of the coronal suture thickening and lamination of the vault

patients who met the following criteria (1) the diagnosis of hydrocephalus of infancy or childhood was confirmed on the basis of cerebral pneumography and other examination (2) the patient had been followed at least four years after initial operation and (3) adequate pre and post operative skull films were available. Twenty five patients met these standards and form the basis for this investigation.

On review post operative skull films were assigned to normal or abnormal groups depending on whether one or more of the following were present. First that there was radiographic evidence for premature closure of one or more sutures. Here complete obliteration of the sutures was required before the diagnosis of stenosis was accepted. Second that there was evidence of lamination of the diploe similar to the description by Du BOUTAL. The appearance was considered by him to represent ghosts of the compact inner and outer table buried in newly deposited bone. A third criterion for abnormality was excessive thickness of the vault. Thickness of the parietal bone was compared to the greatest value measured in films of normal skulls for the same sex and age cohort (HANSMAN) and judged abnormal only if this value was exceeded (Fig 1).

Clinical information was collected from the chart in each case noting the patient's sex, pathologic or etiologic type of hydrocephalus, age at operation, head circumference at operation and age at each shunt revision.

## Results

Results are summarized in Tables 1 and 2. It can be seen that skull films judged abnormal by the criteria enumerated above were more usual than not in this group of patients. Of interest is that lamination was present in

Table 1

*epileptic observations in 25 post-operative patients*

	Present	Absent
Seizures	9	16
Intellectual function	10	15
Attention	14 (56%)	11 (44%)

Table 2

*radiographic correlations in 25 post-operative patients*

	Radiographic group		Total
	Abnormal	Normal	
Communicating hydrocephalus	6	4	10
Non communicating hydrocephalus	8	7	15
Arachnoiditis	7	4	11
Aqueductal stenosis	5	2	7
Congenital malformations	2	5	7
Three or fewer revisions	8	7	15
Four or more revisions	8	4	10

every case in the abnormal category. Pathologic skull thickening and cranio tension were less usual post-operatively and were always accompanied by diploic lamination. This lamination could be observed as early as seven months after shunt operation and where present had always appeared by the twenty fourth post-operative month (Table 1).

Clinically the patients with both normal and abnormal skull films shared many features. No clearly recognizable association with any category of hydrocephalus was apparent in any of the groups represented.

The possibility of a correlation between intermittent shunt obstruction and diploic lamination was of interest. We assumed that cases with poorly functioning shunts would have significantly more revisions than other patients. We counted revisions listed in the patients' charts during four or more years after operation which occurred at 30 day intervals or longer. No association is noted in Table 2 on the basis of intermittent shunt failure.

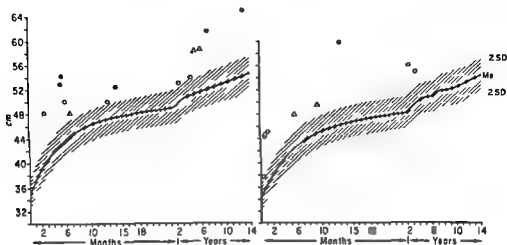


Fig 2 Relationship of skull circumference (at operation) to later post operative radiographic abnormalities in 14 shunted males (left) and in 11 shunted females (right) ○ Normal Δ lamination ● lamination and craniostenosis 48 months later

Head circumference measured at or shortly before operations was compared to sex and age corrected standards for normal (NELLHAUS). It is evident from Fig 2 that patients whose rate of circumferential skull growth greatly exceeded normal were in general likely to present abnormal changes in later films. Small infants appeared to tolerate stretching of the neurocranium without these changes later provided an early shunting operation was performed.

The hypothesis that diploic lamination and its sequelae are caused by stretching of the calvarium is supported by radiographic features exhibited by a six month old girl shunted because of Dandy Walker deformity. Pre-operatively disproportionate enlargement of posterior fossa was observed, while middle and anterior fossae were relatively normal. Changes in the post shunting films 16 months later were limited to the occipital bone where lamination and thickening were visible. The frontal and parietal bones were normal (Fig 3).

Diploic lamination and craniostenosis have been observed radiographically in only one untreated hydrocephalus patient in our experience (Fig 4). These changes were evident at the age of 48 months following a 27 month interval during which the skull circumference had remained stable at 59 cm. This patient had well documented congenital toxoplasmosis.

### Conclusion

In the present group of hydrocephalus patients diversion of cerebrospinal fluid was accomplished through Pudenz Heyer valves with release pressures





Fig 3 Lamination and abnormal thickening confined to the occipital bone in a patient with Dandy Walker deformity 16 months post-operatively



Fig 4 Lamination and frontal thickening 27 months after spontaneous arrest of circumferential skull growth in an untreated patient with hydrocephalus caused by toxoplasmosis

of 60 to 100 cm H<sub>2</sub>O. These patients exhibit abnormal skull films post-operatively more often than not including in some cases premature closure of one or more cranial sutures. The underlying causes in individual cases are not yet clear but may be related to excessive stretching of the neurocranium.

### SUMMARY

Craniosynostosis, abnormal thickening of the skull vault and diploic lamination follow operation for hydrocephalus rather frequently in infants and children in our experience. The occurrence of these complications has no clear association with etiology of hydrocephalus or to the number of shunt revisions required to control intracranial hypertension. Patients are less likely to present these changes if surgical operation takes place early before the cranium has enlarged excessively.

## ZUSAMMENFASSUNG

Kraniosynostose, abnorme Verdickung des Schädeldgewölbes und diploebezugsliche Lamination nach Hydrocephalus Operation werden nach unseren Erfahrungen bei Säuglingen und Kindern ziemlich häufig gefunden. Das Auftreten dieser Komplikationen ist nicht klar zur Ätiologie des Hydrocephalus oder zur Anzahl der Shunt Revisionen, die notwendig sind, um den intracranialen Hochdruck unter Kontrolle zu halten, relativiert. Bei den Patienten, bei denen die Operation frühzeitig vor einer exzessiven Vergrößerung des Schädels vorgenommen wurde, lassen sich derartige Veränderungen weniger wahrscheinlich nachweisen.

## RÉSUMÉ

D'après l'expérience des auteurs, les opérations pour hydrocéphalie chez les nourrissons et les enfants sont souvent suivies d'une cranio-sténose, d'un épaississement anormal de la voûte du crâne et de lamination diploïque. L'apparition de ces complications n'est pas en relation nette avec l'étiologie de l'hydrocéphalie ou avec le nombre des révisions de dérivation nécessaires pour venir à bout de l'hypertension intracrânienne. Ces complications sont vraisemblablement moins fréquentes si l'intervention chirurgicale a lieu avant que le crâne ait beaucoup augmenté de volume.

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## DIE SCHADELFORM FRÜHGEBORENER KINDER

von

H. SCHMIDT und W. HOLTHUSEN

Daß Frühgeborene sich von reifen Neugeborenen nicht nur in Größe und Gewicht sondern auch in den Körperproportionen unterscheiden, ist bekannt und beschrieben.

Der Kopf ist beim Frühgeborenen zwar kleiner als beim reifen normalgewichtigen Neugeborenen (ROBINSON & ROBINSON 1965) er erscheint jedoch relativ zum Körper des Frühgeborenen als zu groß (YIPPO 1931 IWERBECK 1962). Nach den Beobachtungen der beiden letztgenannten Autoren nimmt beim unreifen Neugeborenen die Kopfgröße in den ersten Lebenswochen besonders rasch zu, allerdings mehr durch ein gegenüber der Norm gesteigertes Hirnwachstum als durch eine Größenzunahme des Schädeldaches. Die Nahte sind daher erweitert, die auffallend große Fontanelle ist vorgewölbt (YIPPO).

ROSSIER stellte fest, daß bis zum 6. Lebensmonat noch Unterschiede in der Kopfgröße zwischen dem früh und dem reifgeborenen Kind bestehen. YIPPO sah bis zum Schulalter ALM (1953) noch im Erwachsenenalter Unterschiede in der Körperentwicklung des Frühgeborenen.

Diese zum Teil alten Beobachtungen sind bislang kranio metrisch nicht objektiviert. Nur die Ergebnisse der Ausmessung von Schädeln kurz nach der Geburt

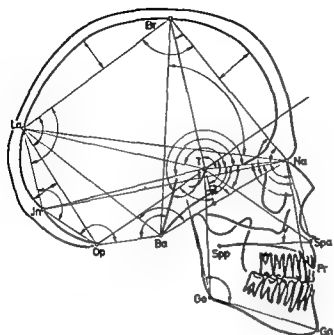


Abb 1 Vermessene Strecken und Winkel am Schädel nach Röntgenaufnahmen im seitlichen Strahlengang (bei Schulkindern)

verstorbener Frühgeborener durch KNAFF BOETTIGER (1959) und publiziert und weiterhin auch Umfangmessungen die nicht nur (unvermeidlich) ungenau sondern für die Beurteilung des Schädelsvolumens auch nicht ausreichen (SCHMID & FILTHUTH 1961)

Danach ergibt es sich als Aufgabe mit kranio-metrischen Methoden der Frage nachzugehen ob — und wenn ja, wie lange — ein Größen oder Formunterschied zwischen Schädeln früh und termingerech geborener Kinder besteht

### Methode und Ergebnisse

Unter Anwendung einer von FENDEL et coll erarbeiteten Methode wurden von BRAUN die Schädelaufnahmen von 105 Frühgeborenen der Tübinger Universitätskinderklinik im Alter von 6 bis 10 Jahren ausgemessen. Die gewonnenen Werte wurden mit den Meßergebnissen von LUTZ (1965) und ZIEGLER (1967) an 162 sagittalen und 154 seitlichen Schädelaufnahmen normaler Kinder der gleichen Altersgruppen (ebenfalls Universitätskinderklinik Tübingen) verglichen.

Die genauen Angaben der Meßpunkte, Meßweiten und die kritische Besprechung der Methodik ist den genannten Arbeiten zu entnehmen. Zur Ori-

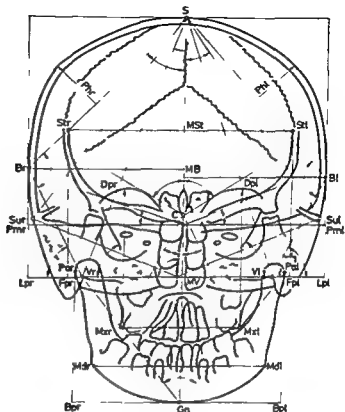


Abb. 2 Vermessene Strecken und Winkel am Schädel nach Röntgenaufnahmen im sagittalen Strahlengang bei Schülkindern

entwertung geben Abb. 1 und 2 eine Auswahl der hauptsächlichsten Meßpunkte, Winkel und Strecken wieder.

Die Vermessung von Schädelaufnahmen nach diesen Meßpunkten ist bei Neugeborenen schwierig. Häufig fehlen die entsprechenden Knochenpunkte. Um der dadurch möglichen größeren Ungenauigkeit zu entgehen, wurde eine weitere Klientel von 17 Frühgeborenen und 72 zum normalen Zeitpunkt geborenen Säuglingen aus dem Kinderkrankenhaus Rothenburgsort nach nur wenigen orientierenden Maßen ausgewertet (Abb. 3 und 4).

Da die Meßpunkte im seitlichen Strahlengang nur eine Auswahl der von Lutz genommenen und besprochenen Maße bedeuten, bedürfen nur die Strecken, die in sagittalen Schädelaufnahmen gemessen wurden, einer Besprechung.

Wie im Seitenbild so wurden auch nach der Sagittalaufnahme im Bereich der Kalotte nur Innenmaße genommen. Die größte Breite liegt individuell in unterschiedlicher Höhe des Schädeldaches. Wenn der Schädel nicht — wie vorwiegend — parietal sondern temporal am breitesten ist, wurden beide Innen-

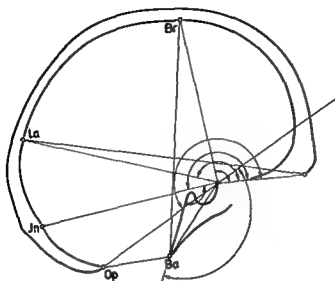


Abb 3 Vermessene Strecken und Winkel am Schädel nach Röntgenaufnahmen im seitlichen Strahlengang (bei Säuglingen)

breiten der Kalotte gemessen. Um weiterhin Breitenmaße der Kalotte in der fixierbaren Höhe zu gewinnen wurde die Kalottenbreite in der Höhe der Orbitadächer und in der Höhe der oberen Pyramidenkanten gemessen. Die höchsten Punkte der Orbitadächer wurden dann verbunden. Die entstandene Strecke wurde nach beiden Seiten bis zur Innenwand der Kalotte verlängert und vermessen. Das gleiche erfolgte mit der Verbindungslinie der höchsten Punkte beider Pyramiden, die bei Säuglingen durch die sehr gut sichtbaren knöchernen Labyrinthkapseln gebildet werden. So ergeben sich 4 Breitenmaße für die Schädelskalotte: 1 die normale parietale Innenbreite, 2 die Innenbreite in der Höhe der Orbitadachpunkte, 3 die maximale temporale Innenbreite, und 4 die Innenbreite in der Höhe der Labyrinthdachpunkte, im folgenden nur mit Nr. 1 bis 4 bezeichnet. Als 5 Breitenmaß wurde für die Schädelbasis die Biventerbreite genommen (ZIEGLER 1967, BRAUN 1970).

Lediglich der groben Höhenorientierung diente ein Maß zwischen dem Scheitelpunkt und der Mandibula sowie zwischen der Orbitadachhöhe (s. Breite 2) und der Höhe der unteren Kontur der vorderen Schneidezähne des Oberkiefers.

Beide Höhenmaße hängen von der Projektion und dabei besonders von der Einstellung des Schädels mit der Deutschen Horizontalen zur Tischebene ab. Die Scheitel-Mandibulahöhe außerdem noch von dem Grad der Mundöffnung während der Aufnahme. Andererseits werden diese Ungenauigkeiten durch die Größe der Varianz statistisch erfaßt und bewertet. Die Höhen werden im folgenden mit I und II bezeichnet.

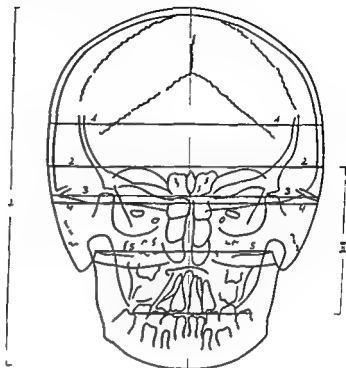


Abb 4 Vermessene Strecken am Schädel nach Röntgenaufnahmen im sagittalen Strahlengang (bei Säuglingen)

Die Tübinger Ergebnisse wurden vom Deutschen Rechenzentrum Darmstadt die Ergebnisse aus Hamburg selbst berechnet

Die Vergleichbarkeit der Varianten wurde nach dem Bartlett Test geprüft Als Grenze wurde eine Irrtumswahrscheinlichkeit von 0,05 gewählt d h die Ergebnisse werden hier nur aufgeführt wenn die Vergleichbarkeit mit 95 % Wahrscheinlichkeit gewahrt ist (ab absoluter Wert über 3,81)

Line gewisse Übersicht über die Disproportionen des Schädels Frühgeborener erhält man schon, wenn man lediglich die erhaltenen Werte des Hirnschädels der Schädelbasis und des Gesichtsschädels nach Länge Höhe und Breite getrennt aufführt In der Tabelle sind die nach dem t Test errechneten p-Werte für die Wahrscheinlichkeit der Differenz eines mittleren Meßwertes der im Frühgeborenen Schädel gewonnen wurde von dem gleichen normalen mittleren Meßwert aufgeführt  $p = 0,02$  bzw  $0,8$  z B heißt daß mit 2 % bzw 98 % Wahrscheinlichkeit keine Abweichung mit 98 % bzw 2 % eine Abweichung von der Norm vorliegt Abolut größere Meßwerte bei Frühgeborenen sind mit einem Plus- kleinere Werte mit einem Minuszeichen versehen Auch hier wird die Wahrscheinlichkeit bei 95 % angenommen wenn p unter 0,05 liegt

Während es die in unterschiedlicher Höhe genommenen Breitenmaße er

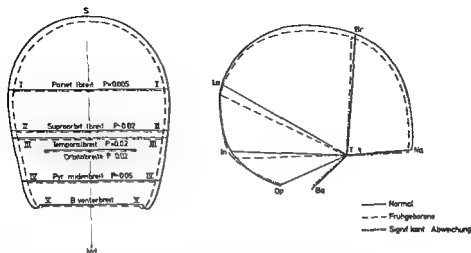


Abb 5 Maßstabgerechte Konstruktion des Schädels im sagittalen und seitlichen Strahlengang nach Messungen an normalen und frühgeborenen Säuglingen

lauben sich ein Bild auch über die Proportionen des Schädels im Sagittalbild zu machen ist dies bei den meisten Meßstrecken des Seitenbildes nicht der Fall. Deshalb werden in dem unteren Teil der Tabelle die Wahrscheinlichkeitswerte für die Abweichungen der Maße am Frühgeborenen Schadel von der Norm wiedergegeben die nach Winkeln am Tuberculum sellae festgestellt sind.

Der Veranschaulichung der Proportions- und Größenunterschiede beim Säugling dient eine Skizze (Abb 5) der Meßstrecken unter Berücksichtigung der Winkel am Tuberculum maßstabgerecht in frontaler Strahlenrichtung auf gezeichnet. Als Bezugspunkt ist T genommen als Bezugsstrecke T Na.

Die Beschreibung der Breitenmaße aller Altersklassen und des Seitenbildes der älteren Kinder ist bei BRAUN nachzulesen. Aus sämtlichen Einzelmaßen lassen sich folgende allgemeine Schlüsse ziehen:

*Bei den Säuglingen* (Eine Vergleichsgruppe Jungen und Mädchen gemeinsam)

1. Der Schadel der Frühgeborenen ist in sämtlichen Dimensionen kleiner als bei zeitgerecht Geborenen.
2. Die Breitenentwicklung ist parietal am stärksten vermindert (Signifikanz über 99,5 % Wahrscheinlichkeit).
3. Eine mit über 95 % Wahrscheinlichkeit nachweisbare verringerte Entwicklung ist bei sämtlichen Breitenmaßen zu verzeichnen.
4. Weiterhin sind mit über 90 % Wahrscheinlichkeit alle Maße verringert die in die Strecken der Schadelbasis einbezogen sind.
5. Proportionsänderungen ergeben sich durch eine relative Verlängerung des Hirnschädels beim Frühgeborenen nach hinten (98 % Wahrscheinlichkeit bei den Winkeln Na T La und Na T J bei zunehmend mangeln



Tabelle  
Streckenmaße bei den Säuglingen

	T—Na	T—Br	T—La	T—Ja	T—O
Norm	n = 71 47.1	n = 70 85.9	n = 72 103.21	n = 69 83.9	n = 71 57.9
Frühgeborene	n = 17 44.8	n = 17 83.6	n = 17 100.23	n = 17 81.8	n = 17 50.1
t Test	2.31	1.31	1.5	1.78	2.96
p etwa	0.02	0.1	0.1	0.2	0.0125
Bartlett Test	0.99	0.18	0.1	0.09	2.96
	2	3	4	5	Orb
Norm	n = 48 112.27	n = 41 111.82	n = 68 99.3	n = 67 74.00	n = 70 80.8
Frühgeborene	n = 13 105.8	n = 9 101.4	n = 10 92.63	n = 17 68.1	n = 16 77.06
t Test	2.24	2.31	1.8	1.9	2.29
p etwa	0.025	0.02	0.075	0.075	0.02
Bartlett Test	0.04	0.64	2.71	3.84	0.00

der Wahrscheinlichkeit einer Verkürzung nach den Strecken T—Br T—La T—J Na—La (siehe auch Skizze Abb. 5)

Bei den älteren Kindern (je zwei Vergleichsgruppen verschiedenen Alters Jungen und Mädchen getrennt): 1. Der Schädel der Frühgeborenen ist nicht in sämtlichen Dimensionen kleiner als bei zeitgerecht Geborenen. 2. Größer sind die Oberkieferlänge (mit Wahrscheinlichkeiten von 97 und über 99.5%) einmal in 4 Gruppen die Prosthionhöhe (99%), einmal in 4 Gruppen die hintere Frontalhöhe (98%), also keine Breitenmaße. 3. Kleiner sind außer ordentlich häufig Breitenmaße meist mit einer Wahrscheinlichkeit von über 99.5%. Längenmaße wenn Strecken der Schädelbasis einbezogen und (Na—La 98%, Na—T und Ba—Op über 99.5%) einige Höhenmaße (in einer einzigen Klasse Mädchen 7/8 Jahr) Mediane Kalottenhöhe (99%), Gesichts- und Obergesichtshöhe (über 99.5%) und einmal in 4 Gruppen die hintere Frontalhöhe (95%). 4. Aus der einzigen Beobachtung einer Winkelvergrößerung von statistischer Bedeutung (Winkel für die Frontalsekante am Tuberculum 98%, Wahrscheinlichkeit) läßt sich kein prinzipieller Schluß auf eine etwa restierende Disproportion des Schädels nach dem Seitenbild ziehen.

Im Vergleich beider Gruppen: 1. Der zu kleine Schädel des Frühgeborenen wird — mit Plus- und Minusdifferenzen — dem Schädel termingerecht gebo-

T—Ba	Na—La	Ba—Br	I	II	I
n = 71	n = 70	n = 71	n = 72	n = 71	n = 71
35.4	144.83	114.7	164	56.83	115.2
n = 17	n = 17	n = 17	n = 17	n = 16	n = 17
32.9	140.67	110.06	158.8	56.4	108.6
9.42	0.86	1.81	1.93	0.62	3.14
0.09	0.2	0.05	0.005	0.25	0.001
1.56	0.05	0.02	0.65	1.39	0.10
Na—T—Br	Na—T—La	Na—T—Jn	Na—T—O	Na—T—Ba	Orb. Dach
n = 71	n = 69	n = 70	n = 71	n = 71	n = 71
80.6	147.5	174.5	201.6	141	164.9
n = 17	n = 17	n = 17	n = 17	n = 17	n = 17
81.8	151	177.6	201.9	141.12	165.2
0.85	2.47	2.11	0.17	0.19	0.41
0.2	0.01	0.0125	0.8	0.9	0.6
0.06	0.37	7.58	0.16	0.3	0.06

rener Kinder an Länge und Höhe fast gleich 2. Der Schädel des Frühgeborenen bleibt bis zum 1. Lebensjahr zu schmal.

### Diskussion

Als Frühgeborene wurden hier nach der Definition der Weltgesundheitsorganisation (WHO) von 1950 Kinder bezeichnet, die bei Geburt 2 500 g oder weniger gewogen haben. Dabei braucht es sich nicht um unreife Kinder zu handeln. Nachdem SCHULTE et coll. (1969) damit rechnen, daß 20 bis 30 % aller Kinder mit einem Geburtsgewicht von unter 2 500 g nicht zu früh geboren, sondern ausgetragen und nur zu leicht sind, wäre für diesen Prozentsatz die Bezeichnung Frühgeborene irreführend.

Diese Feststellung erschwert die Beurteilung der Ergebnisse. Bei den übrigen 70 bis 80 % beobachtet man in den ersten Lebenswochen eine Entwicklung des Schädels, die sonst intrauterin stattfindet.

Nach den Untersuchungen von KNAPP-BOETTCHER nimmt von der 26. bis zur 32. Schwangerschaftswoche das Schädelvolumen besonders rasch zu. In dieser Zeit bleibt die Breitenentwicklung des Hirnschädels gegenüber der Zunahme von Länge und Höhe zurück. Außerdem ändert sich das Verhältnis

Schädelkalotte/Schädelbasis im Sinne eines relativ langsameren Wachstums der Basis

Danach konnte also das bei Frühgeborenen in der ersten Lebenszeit zu beobachtende rasche Kopfwachstum, das nach der Einleitung zu dieser Arbeit durch ein Mißverhältnis in der Größenzunahme von Gehirn und Schädel gekennzeichnet zu sein scheint durchaus noch im Rahmen der normalen sonst sich in utero abspielenden Wachstumsvorgänge bleiben

Nach dem Zeitpunkt jedoch zu dem das zu früh geborene Kind ternun gerecht geboren war bzw. in der das reife mindergewichtige Kind das Gewicht eines Normalgeborenen erreicht hat wächst sein Schädel anders als normaler Weise

Nach YLPRO ruft der Kopf vieler Frühgeborener noch lange den Eindruck eines Puppengesichtes hervor bedingt durch einen relativ großen Hirnschädel mit gewölbter Stirn eine Protrusio bulborum und dicke Wangenfettpolster. Es dürfte sich um den Ausdruck des Mißverhältnisses zwischen Hirn, Augen, Wachstum einerseits und Basis-Orbita-Entwicklung andererseits handeln

Jedenfalls werden YLPROs aus Beobachtung und Erfahrung gewonnenen Schilderungen durch die vorliegenden Meßergebnisse statisch bestätigt. YLPRO sah außerdem Formveränderungen des Kopfes und eine seitliche Abplattung des Schädels. Diese seitliche Abplattung dieses Zurückbleiben in der Breitenentwicklung ist beim Säugling am deutlichsten und beim Kind im Schulalter allein noch mit einem sehr hohen statistischen Wahrscheinlichkeitsgrad festzustellen

Es ist sicher, daß sich aus dieser Feststellung Konsequenzen ergeben. Da die Autoren der Ansicht sind, daß noch zu viele Fragen offen sind, verzichten sie auf weitere Ausführungen und stellen vor jede — etwa gar kausale — Betrachtung folgende Anregungen: 1. Es sollten in der Klinik wieder auch andere Merkmale als nur das Geburtsgewicht zur Charakterisierung eines zu früh geborenen Kindes herangezogen werden. 2. Es sind Untersuchungen des Schädels einer größeren Anzahl von Frühgeborenen mit follow up-studies bis in das Erwachsenenalter hinein durchzuführen. 3. Es sollte das Ziel vieler Untersuchungen mit klinischer, humangenetischer, psychiatrischer und psychologischer Blickrichtung — um nur einige Disziplinen anzusprechen — sein zu erforschen, ob die körperliche Fehlbildung und die Tatsache der Frühgeburt in einem kausalen Verhältnis zueinander stehen oder einem übergeordneten Prinzip unterliegen.

## ZUSAMMENFASSUNG

Nach Meßergebnissen an Röntgenaufnahmen des Schädels von Säuglingen in den ersten 6 Monaten und von Schulkindern im Alter von 6 bis 10 Jahren hat sich der gegenüber

der Norm bei der Geburt zu kleine Schädel des Frühgeborenen bis zum Schulalter sonst fast bis zur Norm ausentwickelt die Breitenentwicklung des Gehirnschädel der Schädelbasis und des Gesichtsschädels schon beim frühgeborenen Säugling am stärksten vermindert bleibt jedoch auch im Schulalter gegenüber der Norm weit zurück

## SUMMARY

Systematic measurements of the skull were performed in roentgenograms in infants up to the age of six months and in school children aged between 6 and 10 years in whom the neonate measurements had seemed to indicate an abnormally small skull. Most dimensions eventually became normal although certain diameters that were particularly diminished in the neonate such as the width of the cranium and of the facial and basal parts of the skull tended to lag behind even when the child attained school age.

## RÉSUMÉ

Des mensurations radiologiques du crâne de nourrissons dans les premiers 6 mois et d'écoliers âgés de 6 à 10 ans ont montré que le crâne du prématuré dont les dimensions sont inférieures à la normale à la naissance se développe pour atteindre des dimensions presque normales à l'âge scolaire. Cependant le développement en largeur du crâne de la base du crâne et de la face qui est déjà chez le nourrisson prématuré le plus diminué reste aussi chez l'enfant d'âge scolaire bien inférieur à la normale.

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## OSSIFICATION OF THE POSTERIOR LONGITUDINAL LIGAMENT

A roentgenologic and clinical investigation

by

M. TAKAHASHI, H. KAWANAMI, M. TOMONAGA and K. KITAMURA

Ossification of the posterior longitudinal ligament has been reported on numerous occasions in the Japanese literature (TERAYAMA et coll 1964, NAKANISHI et coll 1967, OKAMOTO et coll 1967, YANAGI et coll 1967, FURUKA et coll 1968, GOTO et coll 1970) since TSUKIMOTO published the first case in 1960. In the English literature however only a few case reports (OVI et coll 1967, MINAGI & GROVNER 1967) have appeared. The present authors have collected 25 patients with this condition during the past 3 years and the purpose of this communication is to analyze the roentgenologic and clinical findings. Several patients with dorsal and lumbar ossifications are also included since those so far described have always affected the cervical posterior longitudinal ligament (Goto et coll 1970).

*Materials and Methods* The clinical material in this series consists of 25 patients with ossification of the posterior longitudinal ligament. Twenty three had neurologic signs and symptoms attributable to the ossification but 2 patients remained asymptomatic at the time of the investigation. The ages of the patients ranged from 34 to 72. Twenty were males and five were females (Table 1); males thus predominated by a ratio of 4 to 1. The highest incidence occurred

Table 1  
*Age and sex distribution*

Age	Male	Female
30—39	0	1
40—49	7	2
50—59	7	2
60—69	5	0
70—79	1	0

between 40 and 70 years of age. No familial occurrence was evident, no history of trauma was recorded.

### Clinical features

*Symptoms and signs* at the time of the initial visit were the following. Paresthesia was the most common symptom, varying from intermittent sensations of numbness or tingling localized to several digits of one or both upper extremities to extensive and marked anesthesia of the trunk and lower extremities. Twenty-two out of 23 patients complained of paresthesia; this was situated in the upper extremity in 7 patients, in the lower extremity in 11 and 4 patients had symptoms in both the upper and lower extremities. Two patients also had paresthesia of the trunk. Twenty-two patients had motor disturbances. The symptoms varied from clumsiness or weakness of the extremities to incoordination or instability of gait. The motor disturbances were restricted to the upper extremities in one patient; in 7 there was involvement of the lower extremities and 14 patients had symptoms in both the upper and lower limbs.

Seven of the 25 patients complained of headache, neck stiffness or neck pain. Incontinence was present in 4 patients; libido was decreased in 2 patients. Speech disturbance was present in one patient and hiccup in one patient.

The duration of symptoms at the initial examination was the following. Thirteen patients had had symptoms for one to three years before they sought advice. Four patients had a history of less than a year. Six patients gave a history of more than three years, 3 patients had a history of less than ten years and 3 patients of more than ten years. Two patients had no neurologic symptoms referable to the ossification.

*Neurologic findings* were those of segmental neuropathy and myelopathy. The segmental neuropathy was secondary to local involvement of one or more cervical nerve roots. This was manifested by motor disturbances in 15 patients, sensory disturbances in 13 and decreased deep tendon reflexes of the upper













Types		No. of Cases
I		9
		
		
II		10
		
		
III		9
		
		
IV		3
		
		

Fig 1 Type and distribution of ossification

extremity in 7 patients. Five patients had atrophy of the muscles of the arm and 4 patients exhibited fasciculations. The myelopathy was due to compression of the spinal cord. Increased deep tendon reflexes were evident in 21 patients with pathologic reflexes in 13 patients. Fifteen patients exhibited motor disturbances in the lower extremities while sensory disturbances in the lower extremities were present in 14 patients. Seven patients had urinary or fecal incontinence. Neck stiffness was present in 10 patients. Other neurologic findings included Horner's syndrome in 3 patients and dyspnea probably due to phrenic nerve involvement in one patient.

It is interesting to note that no isolated radiculopathy occurred in the series of 23 patients with positive neurologic findings.

Spinal taps were performed on 20 patients and the cerebrospinal fluid findings are given below.

Pressure	Patients
Normal	16
Decreased	4
Queckenstedt's test	
Normal	14
Abnormal	6
Protein content	
Normal	6
Slightly increased (40—100 mg/dl)	9
Markedly increased (> 100 mg/dl)	5





Fig. 2 Types of ossifications in the cervical posterior longitudinal ligament. a) Type II Long rod like ossification posterior and parallel to C2 and C3, a short ossification lies posterior to C4. b) Type III Longitudinally oriented ossification at C3 through C6, small attachments to the upper and lower edges of C4, C5 and C6. c) Type IV Dense ossifications with broad attachments to the posterior surface of the vertebral bodies.

The pressure was abnormally low in 4 patients due to occlusion of the spinal fluid pathways. Queckenstedt's test was abnormal in 6 patients with no rise of fluid levels in 2 and only a sluggish rise in 4 patients. The latter had no rise of the fluid levels on hyperflexion or hyperextension of the neck. In 14 of 20 patients the total protein content was elevated with a slight increase in 9 and a marked increase in 5 patients. There were no other abnormal laboratory findings. The ESR, C-reactive protein, blood chemistry, serum calcium and serum phosphorus were within normal limits.

### Roentgenologic features

*Types of ossification* The ossification may be divided roentgenologically into four types (Fig. 1). The ossifications in type I consist of short bands and are located 1 to 3 mm posterior and parallel to the posterior surfaces of the vertebral bodies. A soft tissue layer usually lies between the posterior surfaces of the vertebral bodies and the ossifications. The 2 to 3 mm thickness of the ossification reduces the spinal canal by 3 to 7 mm anteroposteriorly. The ossifications in type II lie at the posterior surface of the vertebral bodies and merge to form long rod-like ossifications (Fig. 2a). The ossifications in type III have small attach-

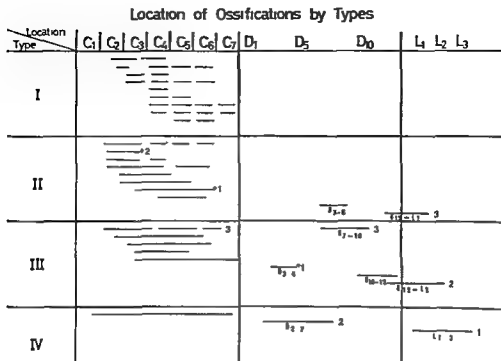


Fig 3 Sites of ossifications by types Ossifications most commonly occur at cervical levels The numbers with a cross indicate patients with both dorsal and lumbar lesions

ments to the upper and lower edges of the vertebral bodies (Fig 2 b) In type IV there is a dense broad direct attachment to the posterior surface of the vertebral bodies and no thin soft tissue layer is present behind the vertebral bodies (Fig 2 c)

It is assumed from the clinical course and neurologic findings in the present material that the lesion progresses from type I to type IV Progression from type II to type III over a four year period occurred in one of the patients and was the only one in whom documentation of the sequential changes in the ossification was possible

**Sites of ossification** The ossification occurs at any cervical level The lesion was at C<sub>4</sub> in 21 of the 23 patients with ossification in the cervical spine Seven patients had ossifications at C<sub>3</sub> and C<sub>5</sub> while in 14 and 11 patients they lay at C<sub>6</sub> and C<sub>2</sub> respectively (Fig 3) Five patients had ossifications within the dorsal or lumbar spinal canal or both, in 3 patients the ossification was observed in both the dorsal and lumbar ligaments

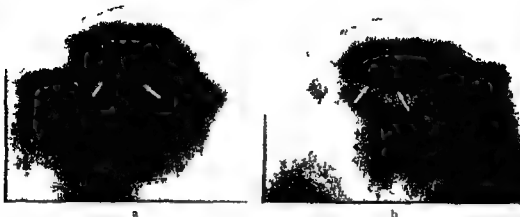


Fig 4 Transverse axial tomography for evaluation of lateral extension and reduced a p diameter a) Dense broad ossification posterior to the vertebral body (←) No soft tissue layer between the ossification and the vertebral body evident b) Ossification extending more to the right (←) Minimal soft tissue layer evident

*Conventional roentgenograms of the spine* The examination of the spine included films in the a p, lateral and oblique projections as well as lateral dynamic views lateral projections were most important as preliminary films A p views failed to disclose the ossification to good advantage unless the lesion was extensive Oblique views were routinely obtained for assessment of encroachment upon the neural foramen There was usually apparent narrowing of the cervical neural foramen which most likely represented superimposition of the ossification The nerve roots were probably compressed at their exit from the spinal cord

Six out of 31 lesions were associated with abnormal curvature of the spine, usually straightening or reversal of the normal curvature

Dynamic views revealed more or less limited movement of the spine in patients with types II, III, and IV ossifications movement of vertebral bodies was usually preserved in the type I patients

*Sagittal and transverse axial tomography* Sagittal tomograms demonstrated the lesions to better advantage (Fig 2 b c) When the findings in the conventional films were questionable tomograms clearly disclosed the lesions Transverse axial tomography was utilized for assessment of lateral extension of the ossifications as well as reduction in the a p diameter of the spinal canal (Fig 4) The clinical signs frequently corresponded to the appearances in the transverse axial tomograms



Fig 5 Gas myelography for evaluation of spatial relationship of the cord to the ossifications and spinal canal. The spinal cord at level of C2 and C3 with minimal cord atrophy. Below C4 the spinal cord is not demonstrated because of compression ( $\leftrightarrow$ )

*Gas and oil myelography.* Gas myelography is more useful than oil myelography since the former makes it possible to evaluate spatial relationships of the spinal cord to the ossification and to the spinal canal. Gas myelography indicated that the subarachnoid space was completely obliterated by the ossification in 7 of 11 patients. It was then possible to evaluate the a-p diameter of the spinal cord and of the spinal canal below or above the ossification (Fig 5). The subarachnoid space was patent in 2 patients in whom the ossification was minimal; another 2 patients had marked atrophy of the spinal cord as outlined by a thin layer of gas.

Oil myelography was performed in selected subjects and usually revealed an anterior extradural defect at the level of the ossification. Lateral views with a horizontal beam direction were essential since a-p views frequently registered fortuitous spinal cord enlargement.

*Relationship of ossification with degenerative osteoarthritis and other ligamentous calcifications.* The incidence of roentgenologic findings of degenerative osteoarthritis is tabulated in Table II. Approximately one half of 31 lesions were associated with degenerative osteoarthritis, but the formation of posterior osteophytes and reduction of the intervertebral spaces were relatively infrequent. Anterior osteophytes were evident in 16 out of 31 lesions. Degenerative osteo-

Table 2

*Associated degenerative osteoarthritis in 31 lesions*

	Same site	Other site
Narrowed intervertebral disc	8	7
Anterior osteophyte	16	17
Posterior osteophyte	9	8
Calcification of paravertebral ligament		
Iliolumbar ligament (2)		
Anterior longitudinal ligament (8)		
Ligamentum nuchae (6)		

Table 3

*Results of surgical treatment in 15 patients*

	No. of patients	Improved	Unchanged	Poor
Laminectomy alone	7	6		1
Laminectomy and foraminotomy	4	3	1	
Laminectomy and section of dentate ligament	3	2		1
Laminectomy and section of dentate ligament and dural plasty	1		1	

arthrosis if present equally involved both the areas with and without ossification. The degenerative changes were minimal to moderate in this series and were considered consistent with the age of the patients. There appears to be no close relationship between the ossification and spondylosis.

**Treatment.** Fifteen of the 23 patients with neurologic disorders were treated by operation (Table 3). Anterior decompression of the spinal cord by Cloward's method was never employed because the ossifications usually covered the posterior surface of more than two vertebral bodies. The dural sac always protruded posteriorly under increased tension through the laminectomy opening and presented no physiologic pulsation. The laminectomy was therefore extended cephalad and caudad until normal pulsation of the dura appeared. Foraminotomy was performed in 4 patients with marked radiculopathy and the dura was

opened in 4 patients the spinal cord was apparently compressed posteriorly by the ossification and the dentate ligaments were detached to mobilize the compressed cord. A hard band like ossification could sometimes be identified by palpation or inspection intra- or extradurally.

There was no death from operation. Quadriplegia appeared postoperatively in 2 patients and in one improved and in the other was associated with minimal difficulty in daily life. Postoperative improvement of neurologic deficits was slow compared with that in cervical spondylosis and the myelopathy in the lower limbs had a tendency to improve more rapidly than segmental neuropathy in the upper extremities. The result of the operation was of course better when the ossifications were confined to one or two levels than when involving several levels, as in type IV.

A patient with minimal neurologic deficits was treated by intermittent neck traction for about four years. The ossification progressed from type II to type III in this period and finally resulted in quadriplegia.

### Discussion

Ossification of the cervical posterior longitudinal ligament occurs in 0.8 to 2.4 per cent of Japanese subjects with cervical spine disorders (ONJI et coll 1967, OKAMOTO et coll 1967, YANAGI et coll 1967, SHIBASAKI & NAGAMATSU 1968). Twenty five instances occurred in the present series of approximately 100 patients with cervical spine diseases admitted for surgery. According to NAKAISHI et coll (1967) however there were 26 patients with ossification of the posterior longitudinal ligament in 83 patients with symptomatic cervical spondylosis among 4 000 outpatients of a neurology clinic. Ossification of the posterior longitudinal ligament is consequently not infrequent in the Japanese. Its ossification however seems to be quite uncommon in non Japanese patients since there have been few reports on this condition from institutions outside Japan. MINAGI & GROVNER (1969) were probably the first to describe similar ossifications in two Caucasian patients; it appeared that the ligamentous ossification had been either overlooked or mistaken for posterior osteophytes associated with degenerative osteoarthritis. CLAWFORD (1967) in a personal communication to ONJI et coll (1967) stated that there were no similar ossifications in a review of roentgenograms of more than 1 000 patients with cervical spine disorders; approximately half of these being of Japanese ancestry in Hawaii.

The reason for the higher incidence of the ossification in the Japanese has not been clarified. There might be some relationships with the habits or modes of living of the Japanese. It is of interest to note that 8 of the 25 patients in this series had ossifications or calcifications in the ligamentum nuchae. In addition,

there were 11 patients who had calcification of the anterior longitudinal ligament. The incidence of ossification in the ligamentum nuchae and anterior longitudinal ligament in these patients is slightly higher than expected. The patient with ossifications may therefore possibly have had a tendency to higher calcium deposit in these ligaments and the soft tissues.

TERAYAMA *et coll.* (1964) reported histologic findings of this condition in a patient who died shortly after surgery. There was an ossification consisting of lamellar structure containing cavities which simulated bone marrow in appearance. A similar bone formation was confirmed at operation in one of the present laminectomy cases. They also stated that there was a thin layer of vascular connective tissue between the ossification and the posterior surface of the vertebral body while in some areas the ossification merged into the posterior wall. The ossifications described by these authors correspond to type II and III of the present classification. Bony attachments at the superior and inferior margins of vertebral bodies in type III correspond to the attachment of the posterior longitudinal ligament to the bodies. Before the ossification involves the level of the intervertebral discs, it is classified roentgenologically as type I. Such an ossification develops into an extensive dense layer which can be classified as type IV. Experience with a patient who progressed from type II to type III over a period of four years and from correlation between the ossification and neurologic findings in other instances suggested that the ossification progresses sequentially from type I through type IV.

Pathogenesis of the ossification in the posterior longitudinal ligament is not clear. TERAYAMA *et coll.* (1964), YAMAMURA *et coll.* (1966) and FURUYA *et coll.* (1968) postulated from pathologic and roentgenologic observations that the primary factor of the ossification is mechanical injury to the posterior longitudinal ligament and to the loose vascular connective tissue behind the vertebral bodies; this is induced by posterior protrusion of degenerated intervertebral discs, and calcification or ossification gradually develops in the posterior longitudinal ligament and the adjoining soft tissues in the reparative process. TAKIMOTO (1960) considered repeated trauma as the predisposing factor in the condition. YANAGI *et coll.* (1967) and YAMAMURA *et coll.* (1966) stressed the higher incidence of calcification in the ligamentum nuchae and anterior longitudinal ligament in these patients, suggesting a general tendency to ossification of the individuals as an important predisposing factor. The present authors also believe that a general tendency to ossification in these subjects plays an important role in calcium deposition and induces ossification in the underlying conditions such as degenerative osteoarthritis and a herniated nucleus pulposus.

Sites of the ossification so far reported have usually been in the cervical posterior longitudinal ligament with few exceptions. OKAMOTO *et coll.* (1967) in

■ series of 21 patients had one patient with an ossification of the posterior longitudinal ligament extending from the cervical down to the upper thoracic area Oht et coll (1967) reported 2 patients with dorsal and lumbar ossifications in their series of 18 patients with ossification of the cervical ligament The present authors had 2 patients with ossifications localized to the dorsal posterior longitudinal ligament while there were 3 patients with ossifications in the dorsal and lumbar areas in addition to the cervical ossification It is presumed that the ossified ligament in the dorsal and lumbar areas may have been overlooked in the patients with cervical ossification However there appears to be definitely a higher incidence of this condition in the cervical areas even though roentgenograms of the dorsal and lumbar spine would usually be obtained less frequently and ossification in the cervical areas might produce neurologic symptoms and signs more frequently

The degree of ossification does not necessarily correspond to the severity of neurologic signs unless marked reduction in the a p diameter of the spinal canal exists Two of the patients had no neurologic symptoms or signs referable to the ossification of the posterior longitudinal ligament

Roentgenologic appearances in this condition are relatively characteristic and the differential diagnosis usually presents no problems In ankylosing spondylitis the ossification is generally situated in the anterior longitudinal ligament and the C-reactive protein and ASLO tests as well as the ESR may be helpful Degenerative osteoarthritis produces calcification around the posterior osteophytes which may simulate ossification in the posterior longitudinal ligament, the calcification in osteoarthritis is not usually arranged in a band like fashion A combination of osteoarthritis and ossification in the ligament may however sometimes exist and possibly the former accelerates the ossification in the ligaments

The surgical treatment of the ossification of the cervical posterior longitudinal ligament is dealt with in the literature by both anterior and posterior approaches the purpose of the operation being to relieve the spinal cord from compression As the ossification usually involves several vertebrae, the posterior approach should perhaps be the procedure of choice Laminectomy should be performed extensively until the normal pulsation of the dura appears an intradural procedure is not essential The present authors emphasize that all procedures should be performed very carefully in order to avoid harmful manipulation of the spinal cord

Conservative treatment such as neck fixation with collar or neck traction should be limited to selected patients with minimal neurologic changes Since the ossification may progress as described, neurologic and roentgenologic controls should always be performed



## SUMMARY

Ossification of the posterior longitudinal ligament in 25 patients is described and the roentgenologic and clinical findings are discussed. Unusual dorsal and lumbar ossifications in 5 patients are also reported. The sites and types of ossification are considered in detail.

## ZUSAMMENFASSUNG

Eine Verknöcherung des hinteren longitudinalen Ligaments bei 25 Patienten wird beschrieben und die roentgenologischen und klinischen Befunde werden diskutiert. Über ungewöhnliche dorsale und lumbare Verknöcherungen bei 5 Patienten wird ebenfalls berichtet. Der Umfang und Typus der Verknöcherung werden im einzelnen behandelt.

## RÉSUMÉ

Les auteurs décrivent l'ossification du ligament vertébral longitudinal postérieur chez 25 patients et examinent les signes radiologiques et cliniques. Ils présentent aussi des ossifications dorsales et lombaires inhabituelles chez 5 sujets. Ils étudient en détail le siège et les types d'ossification.

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## ROENTGENOGRAPHIC CHANGES IN HEADBANGERS

by

J P WILLIAMS G W FOWLER H F PRIBRAM C A DELANEY and C H FISH

Headbanging occurs when a person repeatedly strikes his head against some object. This type of self-inflicted abuse may involve blows to the head with the fist or knees or may consist of striking the head against the floor, wall, bed, etc. This type of behavior is sometimes seen in normal children until age 3 or 4. In mentally retarded or psychiatric patients headbanging may persist until adulthood. Previous investigations in patients with headbanging produced few objective observations and have mainly dealt with the psychological aspects of the disorder.

This paper describes the findings in a large population of headbangers in an institution for the mentally retarded. Approximately 2,000 mentally retarded patients are hospitalized at Fairview State Hospital. A survey revealed that approximately 200 practice some type of self-abuse. The most characteristic form of this behavior was headbanging and occurred in approximately 100. Each patient was investigated by neurologic examination, roentgenographically and with electroencephalography.



Fig. 1 Cauliflower ear in a patient who repeatedly banged the side of his head against his chair. Fragmentation and calcification of the cartilage of the ear.

### Results

As would be anticipated lacerations, hematoma and effusions of the scalp and face were common. Cauliflower ears tended to produce a clinical picture not unlike that in professional boxers. These changes were often demonstrated roentgenographically (Fig. 1).

In rare instances secondary scalp infections occurred and on one occasion progressed to osteomyelitis of the calvarium. In many recurring injuries from headbanging produced massive scar formation and areas of alopecia were typical. In most instances no additional neurologic impairment over that known to be present could be documented. A large number of patients had severe central nervous system abnormalities and consequently new neurologic deficits were difficult to assess and could possibly have been missed. Five patients however were found to have unequivocal changes consisting of pyramidal tract signs and functional deterioration. Spasticity was striking in one patient with Mongolism, a condition in which decreased muscle tone is usually found.

Transient edema and hematomas of periorbital structures were often found but headbanging usually resulted in no injury to the globe itself. In three cases however irreversible lesions of the eye were found. Panophthalmitis developed in one case and led to residual calcification of the eye (Fig. 2). Two additional

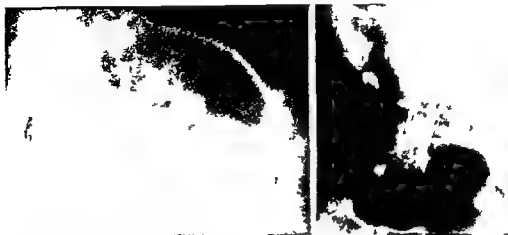


Fig 2 A rounded calcification in the left eye in a chronic headbanger at 16 years of age. No change at age 7.

patients had cataracts a phenomenon previously reported to occur in chronic headbanging (BEMPORAD *et al.* 1968).

Some of the bony changes have been striking particularly when observed over a period of years. They include thickening and irregularity of the calvarium with obliteration of the diploic space and distinct areas of periosteal new bone formation in some cases (Fig 3).

Suture diastasis was demonstrated in several patients and may persist for long periods of time. In the chronically split sutures there is loss of the normal interdigitations such as is seen with long standing increased intracranial pressure (Fig 4). No frank fractures were demonstrated which could be directly attributed to headbanging.

A few of these patients have been studied with encephalography. None had ventricular enlargement even patients with suture diastasis.

Electroencephalographic abnormalities were demonstrated in a number of cases but only those patients with serial recordings will be described. After excluding all cases in which EEG changes could conceivably result from progressive neurologic disease or uncontrolled seizures six patients showed persistent EEG abnormalities consisting of diffuse or focal slow wave activity. In an additional two cases of Mongolism progressive EEG changes were noted but these could be attributed to the early development of Alzheimer's disease known to occur in Down's syndrome. Although few in number the electroencephalographic abnormalities found in headbangers were surprising since previous



Fig. 3 a) Lateral view of the skull. Marked thickening of the calvarium on both sides of the coronal suture, which was the primary area of abuse in this patient who banged his head against the floor and wall. b) A distinct layer of periosteal bone formation much like the in completely calcified cephalohematoma seen in *nes* horns.

observers had either not mentioned this occurrence or had reported normal EEG (KRAVITZ 1960). However, previous reports dealt with relatively minor degrees of headbanging and in children essentially free of neurologic disease (GIBBS 1970).

### Discussion

Investigations in headbanging have for the most part dealt with psychodynamic factors and have not included neurologic, roentgenographic, or electroencephalographic descriptions of their patients. The condition which offers the greatest similarity is that of the encephalopathy described in professional boxers. Although clinically similar, none of the encephalographic changes in boxers was demonstrated in our patients with headbanging (ISHERWOOD 1966, MAWDSLEY 1963, SPILLANE 1962). In most instances, an explanation of merit could be offered for the roentgenographic abnormalities. For example, thickening of the calvarium was felt to be due to repeated subperiosteal hemorrhage with resulting calcification. These lesions in many respects are similar to those found in cephalohematomas of newborns. The pathogenesis of the diastatic sutures, however, remains unknown. Several explanations for this curious phenomenon have been offered, but none is entirely convincing. The loss of normal bony interdigitations of the suture lines is seen with increased intracranial pressure. But in none of our patients was there marked ventricular enlargement, nor was papilledema present. Development of leptomeningeal cysts has also been proposed as a possible explanation for the chronic suture separations (SCHIECHTER 1970), but careful



Fig 4 Widely diastatic sagittal suture and slight separation of the lambdoid suture in this severe headbanger. Loss of the normal bony interdigitations on the margins of the parietal bones along the sagittal suture. This patient had a sunken area externally along the course of his sagittal suture and no ventricular enlargement.

search for these cysts fail to support this explanation. In fact, the characteristics of the separation were quite dissimilar to those found in leptomeningeal cysts.

Although all of the patients in this investigation had as a common denominator varying degrees of mental retardation, they represent a broad spectrum of neurologic disease and no common etiologic factor was apparent. Similarly, the severity of retardation did not seem to be related to the severity of either the roentgenographic or electroencephalographic changes.

In one case (Fig 3) in which both clinically and roentgenographically the changes were very marked, empirical psychosurgical treatment was attempted by bilateral amygdalotomy, with considerable beneficial results. In other cases of less severity, psychosurgery and electroconvulsive treatment did not achieve significant or lasting results.

In no case could a fatal outcome be ascribed to headbanging. In fact, regardless of severity or treatment, the syndrome tended to "burn-out" by early adulthood in much the same way as other forms of hyperkinetic behavior in mentally retarded persons.

## SUMMARY

One hundred severe headbangers were investigated neurologically, roentgenologically, and electroencephalographically in a hospital for the mentally retarded. Clinical and electroencephalographic evidence of injury to the central nervous system attributable to headbanging was found. Soft tissue hematomas, cauliflower ears, calcification of the eye thick

ening of the calvarium diastasis of the cranial sutures and a hematoma of the corpus callosum were demonstrated roentgenographically. A brief discussion of incidence etiology and therapy is included.

## ZUSAMMENFASSUNG

Hundert schwere Kopfstosser wurden neurologisch radiologisch und encephalographisch in einem Krankenhaus für mental Retardierte untersucht. Es wurden klinische und encephalographische Zeichen für Schaden des Zentralnervensystems die auf Stösse mit dem Kopf zurückgeführt werden können gefunden. Hamatome der weichen Gewebe Blumen kohlernen Verkalkung des Auges Verdickung des Schädeldachs Diastase der Schädel suturen und ein Hamatom des Corpus callosum wurden gefunden bzw. roentgenologisch nachgewiesen. Der Umfang die Ätiologie und die Therapie werden kurz besprochen.

## RÉSUMÉ

Les auteurs ont fait une étude neurologique radiologique et électroencephalographique dans un hôpital pour retardés mentaux de cent enfants qui se cognent la tête fortement (headbangers). Ils ont trouvé des signes cliniques et électroencephalographiques de lésions du système nerveux central qui peuvent être attribuées au cognement de tête. Ils ont mis en évidence radiographiquement des hématomes des parties molles des oreilles en chou fleurs des calcifications oculaires un épaississement de la voûte du crâne un diastasis des sutures crâniennes et un hématome du corps calleux. Les auteurs terminent par une brève étude de la fréquence de l'étiologie et du traitement de cet état.

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## ANGIOGRAPHY





## PHLÉBOGRAPHIE MÉSENCEPHALIQUE DANS LES TUMEURS DU TRONC CÉRÉBRAL

par

J P BRAUN et A WAGLENHEIM

L'étude angiographique des veines mésentéphaliques postérieures apporte souvent des critères intéressants dans le diagnostic des masses expansives de la région du tronc cérébral. La veine mésentéphalique postérieure prend son origine dans la région antérieure ou latérale du pedoncule cérébral, contourne le tronc cérébral et se jette dans la veine de Galien, en drainant les régions pontique supérieure et mésentéphalique.

Sur une incidence de face a p le repérage des veines mésentéphaliques postérieures est parfois difficile à cause de la finesse du calibre de ce vaisseau mais on peut très souvent l'identifier. Son trajet prend une direction ascendante médiane parallèle à celui du segment postérieur de la veine basilaire (Fig 1). Les deux veines présentent un aspect en "V" renversé déjà décrit par HUANG.

Sur une incidence de profil la veine mésentéphalique postérieure présente un trajet rectiligne à direction ascendante postérieure parallèle au segment postérieur de la veine basilaire. Ces veines sont souvent superposées et mal individualisées. Lorsqu'elles sont opacifiées des deux côtés, les 4 vaisseaux présentent une image de superposition plus ou moins enchevêtrée (Fig 1). Lorsque le drainage veineux basilaire se fait à travers la veine anastomotique mésentéphalique latérale



Fig 1 a) Phlebogramme de profil Topographie normale de veine mesencephalique postérieure (→) qui croise le trajet postérieur de la veine basilaire b) Phlebogramme de face La veine mesencephalique postérieure (→) chemine parallèlement et en dessous de la veine basilaire



Fig 2 Schema et photographie de la pièce de dissection de région mesencephalique 1 Veine mesencephalique postérieure 2 Veine précentrale 3 Veine anastomotique mesencephalique latérale 4 Veine de Galien 5 Tubercules quadrijumeaux 6 Vermis cerebelleux

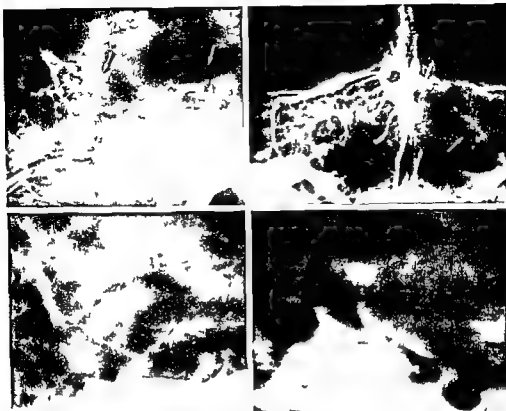


Fig 3 Glome du tronc cérébral supérieur Phlebogramme Dissociation des 3 troncs veineux basilaire et mesencéphalique postérieur (→) Profil système communicant postérieur est comprimé La jonction de la veine communicante postérieure et la veine mesencéphalique postérieure est fortement comprimée et abaissée Refoulement de la veine précentrale Face Dilatation et déformation du système communicant postérieur Tension de la veine mesencéphalique latérale gauche

rale la veine mesencéphalique postérieure est mieux visible Nous avons eu l'occasion de dissequer un cas semblable représenté sur la Fig 2 La veine chemine en dehors du mesencéphale en avant de la veine précentrale Grâce à la connaissance de la radioanatomie veineuse il est possible de reconnaître des altérations suffisamment importantes pour orienter le diagnostic de masse expansive de la région mesencéphalique

Nous avons publié dans une monographie (WACKENHEIM & BRAUN 1970) l'intérêt du diagnostic phlebographique et apportons ici les conclusions de 10 observations complémentaires (Fig 3—6) L'étude de ces 10 phlebogrammes nous permet de faire les commentaires suivants

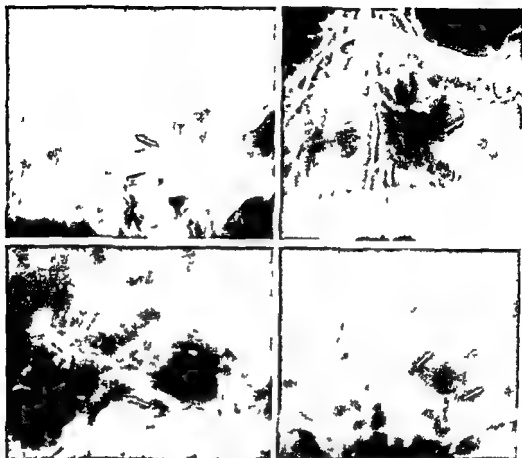


Fig. 4. Gliome examiné à 8 ans d'intervalle chez un garçon de 15 ans. Phlébogramme. Profil. Leger refluxement de la veine precentrale. Refluxement plus prononcé de la mesencephalique laterale. Ces veines presentent un trajet a concavité antérieure. Dissociation des troncs veineux basilaire et mesencephalique postérieur. Face. Forte deviation laterale de la veine mesencephalique laterale et de la veine de Dandy surtout a gauche. Hypertrophie et irregularite de la veine communicante postérieure du cote gauche. Phlébogramme 2 ans apres (en bas). Profil. Refoulement de la veine precentrale et de la veine mesencephalique laterale qui presentent des trajets rigides. Face. La veine mesencephalique laterale droite est comprimee filiforme. Elle est dilatee du cote gauche. La veine communicante postérieure est irreguliere. Remarquez du cote gauche une dissociation de la veine basale par rapport a la veine mesencephalique postérieure.

*Incidence de face :* (1) Les veines mesencephaliques posterieures sont deplacées vers l'exterieur et vers le haut (Fig. 5). (2) Les alterations du systeme veineux communicant postérieur sont frequentes : hypertrophie et irregularite du calibre (Fig. 3, 4) ou deplacement (Fig. 6) et interruption du trajet.

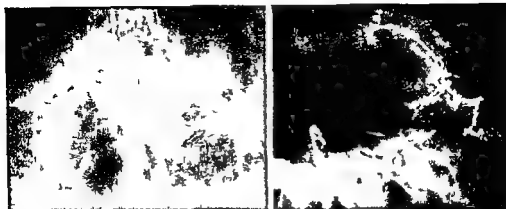


Fig 5 Glomé de la protuberance Phlebogramme Profil : Légère dissociation des veines mésentéphaliques postérieures Rectitude et refoulement des veines ponto-mésentéphaliques La veine mésentéphalique latérale présente un trajet à concavité antérieure Face Déviation externe des veines mésentéphaliques latérale et postérieure du côté gauche

*Incidence de profil* (1) Abaissement du segment antérieur de la veine mésentéphalique postérieure (Fig 3 6) Ce segment peut être absent congénitalement dans ce cas il faut tenir compte des veines pontiques dont la tension et la rectitude du trajet remplacent le signe de la veine mésentéphalique postérieure (Fig 6) (2) Irregularité du trajet des veines mésentéphaliques postérieures dans le segment postérieur (3) Dissociation des segments postérieurs de la veine basale et de la veine mésentéphalique postérieure (Fig 3 4 6)

Dans ces communications nous n'avons pas tenu compte des modifications parfois importantes et caractéristiques des veines pontiques ponto mésentéphaliques mésentéphaliques latérales precentrale et basilaire qui ont fait l'objet d'études antérieures Nous rappelons néanmoins que nous retrouvons le déplacement surtout postérieur de la veine precentrale décrit par HUANG Une tumeur du tronc cérébral peut soulever les veines de la région ponto mésentéphalique par la topographie parfois excentrique de l'expansion tumorale Les Fig 3 et 6 illustrent ce propos Le déplacement de ces veines semble paradoxal sur une incidence de profil comme si la pression latérale pouvait soit soulever soit abaisser ces veines

Dans cette communication l'iconographie de 4 observations seulement a pu être retenue

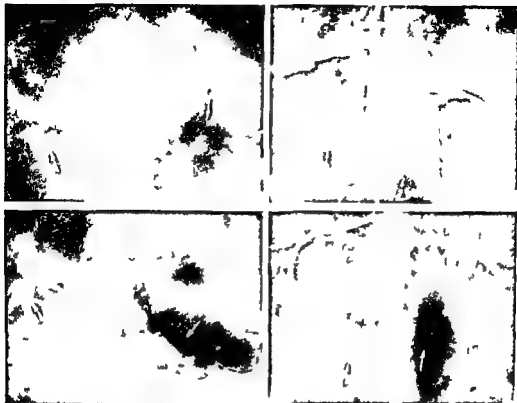


Fig 6 Gliome de la protuberance examiné à 5 ans d'intervalle. Fil bogramme. Profil. Le segment postérieur de la veine basilaire est un peu irrégulier et mal opacifié. Les veines du système communicant postérieur sont agglutinées. La veine précentrale est nettement refoulée vers l'arrière. La veine mésoencéphalique latérale paraît normale. Face. Soulèvement du système communicant postérieur. Fil bogramme 5 ans après (en bas). Profil. Dans la région mésoencéphalique supérieure on distingue deux troncs veineux nettement distincts. La veine précentrale est maintenant refoulée vers le haut et l'arrière. Face. Le système communicant postérieur est soulevé et une capillarographie apparaît sur la ligne médiane.

## RÉSUMÉ

Les auteurs rapportent une étude phlébographique basée sur 10 observations vérifiées de tumeur du tronc cérébral. Les altérations veineuses sont décrites et particulièrement celles des veines mésoencéphaliques postérieures. Ils estiment que ces modifications pathologiques veineuses sont intéressantes à connaître pour la localisation angiographique des masses expansives de la région ponto-pédonculaire.

## SUMMARY

The phlebographic findings in 10 cases of tumours of the brain stem are reported. Particular attention was paid to the appearances of the posterior mesencephalic veins. Changes in the veins appeared to contribute much to the identification of tumours of the pontopedicular region.

## ZUSAMMENFASSUNG

Das phlebographische Bild von 10 Fällen von Tumoren des Hirnstammes wird analysiert. Besondere Aufmerksamkeit wird auf das Röntgenbild der hinteren Venen des Mittelhirnes gerichtet. Es zeigte sich, dass das phlebographische Bild von grossem Nutzen ist, wenn es notwendig ist, Tumoren der ponto pedikularen Region zu lokalisieren.

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## ANGIOGRAPHIC ESTIMATION OF THE SIZE AND SITE OF THE LATERAL AND FOURTH VENTRICLES

by

S BRIC and Z WOJCIEK

The range of indications for the angiographic investigation of the carotid and vertebral arterial systems has extended considerably in recent years (MUSZYŃSKI 1968). The examination is not time consuming, technically easy to perform and the patients feel comparatively more comfortable than during encephalography (BRIC 1968, DECKER 1960, GALLOWAY et coll. 1964).

The routine use of angiography for clinical purposes raises the question whether the size and position of the cerebral ventricles can be assessed exclusively from carotid and vertebral angiographic examinations. It would certainly be of value to establish the correct roentgenologic diagnosis. In hydrocephalus the course of the pericallosal artery is more obtuse and the normal irregularity of its course is smoothed out; the Sylvian group of vessels is elevated and displaced forwards with considerable increase in the angle made with the base of the skull. The observations of the present authors as well as the literature indicate, however, that the degree of vascular changes is frequently not directly proportional to the amount of ventricular dilatation as revealed by encephalography. The enlargement of the lateral ventricles is often relatively marked before a significant

vascular change becomes apparent. A comparison of encephalograms with angiograms obtained in the same patient has failed to prove the practical possibility of determining the true size of the lateral ventricles. Carotid angiography in hydrocephalus usually reveals that the pericallosal artery is not only raised but also displaced medially, thus the upper margin of the body of the lateral ventricle cannot be defined by the course of this artery.

Some authors have stated that hydrocephalus may be detected by vertebral angiography if arteries such as the posterior cerebral artery are straightened (GALLOWAY et coll 1964, FRIEDENBERG et coll 1960). Such changes are however late features. In an earlier stage of hydrocephalus attention should be paid to the centrally situated vessels that are closely connected with the lateral ventricles: these are the medial and lateral choroid arteries and the posterior pericallosal artery. An apparent gap between the posterior pericallosal artery curving around the corpus callosum and the lateral choroid arteries behind the thalamus within the choroid fissure should give some indication of the size of the posterior part of the lateral ventricle.

The authors attach great importance to the position of the veins as demonstrated in the half axial view in the supine position in the assessment of the ventricular size. Thus the striothalamic vein lies for most of its course in the lateral wall of the lateral ventricle so that a change in the size or shape of the ventricle is faithfully reflected in the course and configuration of the striothalamic vein.

The anterior part of the lateral ventricle may be traced in the lateral projection with the aid of the small branches of the vena septi pellucidi. Several small veins collect blood from the internal surface of the anterior parts of the ventricles: the genu corporis callosi and the caput nuclei caudati. Small vessels draining blood from the medial part of the lateral ventricle join the striothalamic vein which runs in the wall of the lateral ventricle forwards in the sulcus separating the thalamus from the caput nuclei caudati. Both veins emerge through the interventricular foramen and become the paired internal cerebral veins that run above the third ventricle in the tela choroidea. These veins join the cerebri magna vein which is supplied by a few tributaries, and subsequently run into the sinus rectus.

Small tributaries of the striothalamic and septi pellucidi veins appear in the phlebograms at their origin in the shape of the letter T as they join its horizontal arms: a true outline of the upper and anterior margin of the lateral ventricles is obtained: their lower margins are situated at the junction in the interventricular foramen of the veins mentioned. While the ventricles become larger the veins straighten out according to the ventricular size. An understanding of the normal and pathologic variants in the shape of the ventricles at cerebral angiography may sometimes render encephalographic procedures unnecessary.

The position of the fourth ventricle in normal as well as in pathologic conditions may be evident from the vertebral angiogram. The inferior posterior cerebellar artery arises from the vertebral artery, runs through the cisterna medullaris and then laterally to the medulla before changing its direction downwards. The medial branch then runs upwards along the inner side of the cerebellar tonsil to the roof of the fourth ventricle. Small branches course to the choroid plexus of the fourth ventricle while the last part of the medial branch runs across the posterior part of the tonsil in a dorsomedial direction (PRETERS 1968). As to the location, GREITZ & SJÖGREN (1963) have divided this vessel into three parts, the cisternal, medullary and choroidal parts. The situation of the latter is of considerable value in establishing the correct position of the fourth ventricle. A reliable landmark for the localization of the fourth ventricle appears to be the point of Twining. This normally lies just about in the middle of the fourth ventricle and the beginning of the arch of the choroidal part of the inferior posterior cerebellar artery normally lies less than 1 cm below Twining's point. This is a reliable sign to indicate displacement of the fourth ventricle posteriorly, as occurs when a tumour is present in the pons or its immediate surroundings. A correct diagnosis from vertebral angiography alone is most satisfactory for such an examination in patients with increased intracranial pressure and often in a poor general condition involves less risk than encephalography. The diagnosis of expanding lesions situated in the pons and cerebellar hemispheres by vertebral angiography is of much practical significance.

## SUMMARY

The angiographic investigation of the carotid and vertebral arterial systems as a means of determining the position and size of the ventricles is discussed. Attention is drawn to the value of being able to localize the fourth ventricle by angiography.

## ZUSAMMENFASSUNG

Es wird beschrieben wie man mittels Angiographie der A. carotis und der A. vertebralis möglich ist die Lage und Grösse der Hirnventrikel zu bestimmen. Der Wert der Angiographie um die Lage des vierten Ventrikels zu bestimmen wird betont.

## RÉSUMÉ

Les auteurs décrivent l'utilisation de l'angiographie carotidienne et vertébrale pour déterminer la position et les dimensions des ventricules. Ils attirent l'attention sur l'intérêt de pouvoir localiser le quatrième ventricule en utilisant l'angiographie.

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## POSTERIOR INFERIOR CEREBELLAR ARTERY IN FOURTH VENTRICULAR DILATATION

by

J B BURNS, J C HOFFMAN and J R BRYLSKI

The posterior inferior cerebellar artery (PICA) is of considerable importance in the angiographic evaluation of posterior fossa lesions. The close proximity of this vessel to the fourth ventricle and its relationship to the cerebellum permit radiographic evaluation of axial shift resulting from mass lesions within the posterior fossa (GREITZ & SJOGREN 1963 WOLF et coll 1962). Changes in the configuration of the PICA may also be the result of congenital anomalies involving either the brain stem or the ventricular system (GREIFUSS & PUCKETT 1969 HUANG et coll 1968 JUHL & WESENBERG 1966). In view of the strategic location of the fourth ventricle it has been postulated that alteration of the PICA could arise from an acquired increase in ventricular size. It has also been speculated that these changes could simulate the radiographic findings attributed to mass or congenital lesions (GREIFUSS & PUCKETT).

It is the purpose of this paper to describe the effects of acquired enlargement of the fourth ventricle on the PICA. The angiographic findings characterizing increased fourth ventricular size and their potential confusion with other lesions of the posterior fossa will be discussed. Cases illustrating pertinent angiographic abnormalities will be presented.



Fig 1 a) Lateral vertebral angiography demonstrating the normal course of the PICA. The cephalic curve of the choroid loop is indicated by the arrow. b) Pneumographic autotomography illustrating the normal configuration of the fourth ventricle. The arrow points to the ventricular wall which is formed by the ependymal surface of the posterior medullary velum. The choroid loop of the PICA parallels this convexity on the lateral projection.



Fig 2 a) Ap vertebral angiography demonstrating the normal relationship of opposing choroid segments (→). Separation of these vessels at this point is of concern when over 5 millimeter and generally abnormal when over 1 centimeter. b) Pneumographic demonstration of the fourth ventricle on p.a. projection. Air is retained within the fourth ventricle by the confluence of the anterior and posterior medullary velum at the apex. The choroid loop crosses the ventricular silhouette (→).



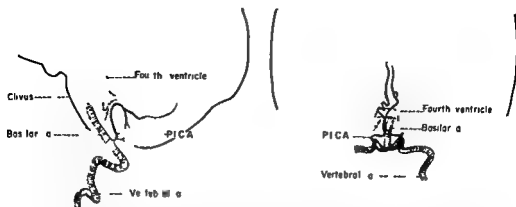


Fig 3 Diagram of the normal fourth ventricle and its relationship to the PICA. The choroid loop (→) is separated from the ventricle by the posterior medullary velum. Distortion of this membranous septum would exert a direct influence on the choroid vasculature. In the frontal view the choroid loop is illustrated as it crosses the air-filled fourth ventricle (↔).

**Normal anatomy.** The PICA commonly arises from the distal two centimeters of the vertebral artery. Occasionally it originates from the base of the basilar artery. After circumventing the brain stem, the vessel extends rostrally over the posterior medullary velum. Its choroid loop (Fig 1 a) parallels the cephalic curve of the posterior ventricular wall (Fig 1 b). Terminal branches extend over the inferior surface of the vermis and cerebellum. Opposing choroid segments approach midline on the a.p. projection (Fig 2 a). Separation of the choroid loops was previously reported to rarely exceed four millimeters (Wolf et coll 1962). In our experience these loops have been separated by as much as 1 centimeter in patients with no enlargement of the fourth ventricle. A diagrammatic illustration of the normal ventricle and its relationship to the PICA emphasizes the intimate relationship of the posterior medullary velum and the choroid loop (Fig 3). Distortion of the ventricle in this area would exert a direct influence on the choroid vasculature.

### Case reports

**Case 1 Occult communicating hydrocephalus** (Adams et coll 1965). A 71 year old white female was hospitalized with a twelve month history of progressive mental deterioration. Her symptoms were preceded by a non-specific febrile illness. Physical examination revealed a generalized impairment of mentation without evidence of localized neurologic deficit. Bilateral papilledema was noted. Skull roentgenograms and a rectilinear brain scan were unremarkable. Carotid angiography demonstrated severe symmetrical enlargement of the lateral ventricles. Vertebral angiography was performed to determine the etiology of the hydrocephalus. Ventral displacement of the basilar artery in association with separation of

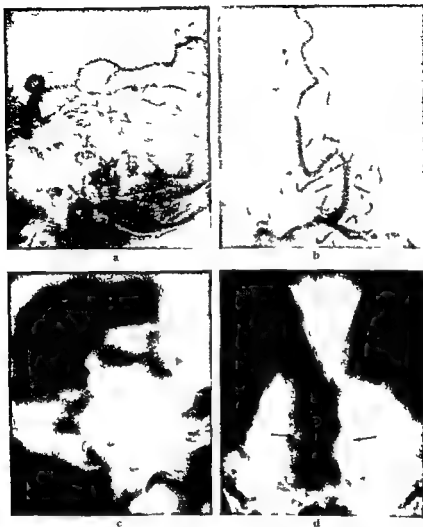


Fig 4 Case 1 a) lateral and b) ap views of subtraction angiography. The appearance of the PICA is normal ventral displacement of the basilar artery. The choroid loops are abnormally separated by 10 mm ( $\rightarrow$ ) the vermis branches of the PICA in normal position ( $\leftrightarrow$ ) c) lateral and d) pa view of ventriculography. Moderate increase in fourth ventricular width particularly prominent ( $\rightarrow$ ) e) Diagram illustrating the abnormal separation of opposing choroid loops ( $\rightarrow$ ) and their relationship to the increase in fourth ventricular width.

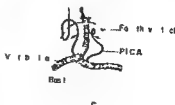




Fig 5 Case 2 Vertebral angiography a) Lateral view Marked stretching and inferior displacement of the choroid loops ( $\rightarrow$ ) Ventrocaudal shift of the tonsillar loop ( $\rightarrow$ ) The vermis branches of the PICA elevated and stretched ( $\rightarrow$ ) b) A p view A 13 millimeter separation of the choroid loop ( $\rightarrow$ ) as well as severe separation of the vermis and tonsillar segments of the PICA ( $\rightarrow$ )

the choroid loops suggested a mass within the cerebellar vermis (Fig 4 a b) The ventriculography failed to substantiate displacement of posterior fossa contents by tumor It did demonstrate an enlarged fourth ventricle which retained its normal configuration (Fig 4 c d) Subarachnoid air did not extend beyond the tentorium RISA cisternography (WAGNER 1968) was consistent with an obstruction of the subarachnoid space Following an atrio ventricular shunt the patient improved considerably The abnormal separation of opposing choroid loops and their relationship to an increase in fourth ventricular width are illustrated in Fig 4 e

*Case 2 Non communicating hydrocephalus* A 20 year old Negro male was hospitalized with a six day history of headache diplopia and somnolence One year before admission he had had mumps meningoencephalitis from which he completely recovered Papilledema was demonstrated on physical examination Sellar changes suggesting increased intracranial pressure were noted on the skull roentgenograms Carotid angiography demonstrated severe symmetrical ventricular enlargement Changes on vertebral angiography involving the PICA were consistent with a fourth ventricular or vermis tumor (Fig 5) A 13 millimeter separation of the opposing choroid loops was demonstrated Ventriculography revealed severe generalized ventricular enlargement Cystic displacement of the posterior medullary velum within the cisterna magna was noted (Fig 6) The effects of these changes are diagrammatically illustrated in Fig 7 The patient improved following an atrio ventricular shunt



Fig 6 Case 2 Ventriculography a) Lateral view Marked generalized fourth ventricular enlargement Cystic protrusion of the posterior medullary velum into the cisterna magna ( $\rightarrow$ ) Air was retained at the foramen magnum and at the medial margins of the cerebellar hemispheres b) Pa view Severe enlargement of the fourth ventricle is demonstrated ( $\rightarrow$ ) The mass like effect of the cystic protrusion of the medullary velum is also illustrated ( $\leftrightarrow$ )

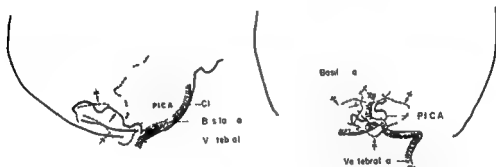


Fig 7 Case 2 Diagram relating the alteration of the choroid loop to the ventricular changes ( $\rightarrow$ ) Distortion of the PICA resulted from the mass like displacement of the posterior medullary velum into the cisterna magna ( $\leftrightarrow$ )

### Discussion

Alteration of the PICA as a result of an increase in the width of the fourth ventricle is suggested by the separation of opposing choroid segments of this vessel on the a p projection at vertebral angiography. The paucity of associated angiographic involvement of the PICA demonstrated in Case 1 emphasizes the diagnostic need for bilateral demonstration of these vessels. It is anticipated that an erroneous impression of brain stem shift on the basis of lateral choroid displacement could occur if only one side is contrast filled.

As expansion of the fourth ventricle progresses added distortion of the PICA will be apparent. The additional angiographic findings in Case 2 resulted from the concurrent mass like extension of the posterior medullary velum into the

cisterna magna. Angiographic distortion of the fourth ventricle was indistinguishable from the changes attributed to mass lesions either within the fourth ventricle or the vermis of the cerebellum. It is conceivable that similar changes could be the result of congenital obstruction of the fourth ventricle, although associated anomalies of the cerebellum and related vasculature suggests a potential means of differentiation.

### Acknowledgements

The authors are grateful to Grover H. Hogan, Medical Artist, and Robert H. Beveridge, Medical Photographer, for their efforts in the production of this paper. This was in part supported by Public Health Service Grant No. 7T1 05494-08.

### SUMMARY

Angiographic alteration of the posterior inferior cerebellar artery as a result of acquired fourth ventricular enlargement may simulate mass or congenital lesions within the posterior fossa. Because there is a spectrum wherein both the normal and pathologic lie, separation of the choroid loops can only be of diagnostic value when there is angiographic evidence of lateral ventricular enlargement.

### ZUSAMMENFASSUNG

Angiographische Änderungen der Arteria cerebellaris posterior als Folge einer erworbenen Vergrößerung des vierten Ventrikels mögen grobe oder angeborene Veränderungen innerhalb der Fossa posterior vortauschen. Da es ein Spektrum innerhalb der normalen und pathologischen Lage gibt, ist die Auftrennung der choroidalen Schlingen nur von diagnostischem Wert, wenn der angiographische Nachweis einer Vergrößerung des lateralen Ventrikels vorliegt.

### RÉSUMÉ

Les modifications de l'aspect angiographique de l'artère cérébelleuse postérieure et inférieure dues à une dilatation acquise du quatrième ventricule peuvent simuler une tumeur ou des lésions congénitales de la fosse postérieure. Étant donné la similitude possible entre les aspects normaux et pathologiques, l'écartement des boucles choroidiennes ne peut avoir une signification diagnostique que quand il y a des signes angiographiques de dilatation des ventricules latéraux.

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## ETUDE MICROANGIOGRAPHIQUE DU CORTEX CÉRÉBRAL DANS LE SYNDROME DE LA DÉMENCE SENILE

par

M. COLLARD

L'incidence des artéropathies dans divers syndromes psychiques du sujet âgé et plus particulièrement dans la détérioration ou la confusion mentale est difficile à évaluer quantitativement.

La moitié environ des démences seniles est mixte : elles résultent à la fois de lésions dégénératives et de lésions vasculaires se caractérisant par des foyers de démyélinisation de la substance blanche et des ramollissements corticaux et sous-corticaux.

Dans l'ischémie cérébrale d'origine tronculaire résultant d'une pathologie intéressant les artères cérébrales principales les lésions sont engendrées par une modification de facteurs hydrodynamiques. Par contre, dans l'ischémie lacunaire à des lésions intéressant les artérols corticales, le processus est plus diffus et ne présente pas de localisation préférentielle dans le territoire pariéto-temporo-occipital.

Nous avons étudié 32 patients présentant un syndrome de démence senile à différents stades évolutifs et nous avons comparé la clinique aux modifications artérielles mises en évidence par l'angiographie carotidienne et vertébrale *in vivo* et aux altérations artérielles et artériolo-capillaires démontrées par la microangiographie.

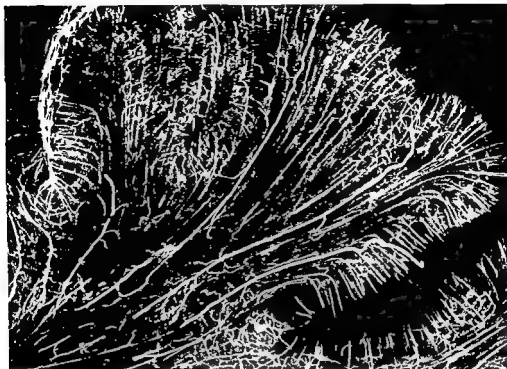


Fig 1 Microangiographie d'une circonvolution cérébrale. Agrandissement  $\times 6$ . Démonstration du réseau artériolaire avec opacification des veines marginales. Atheromase artériolaire diffuse.



Fig 2 Microangiographie. Agrandissement  $\times 10$ . Des artérioles axiales normales alternent avec des artérioles présentant des lésions de surcharge.



## ETUDE MICROANGIOGRAPHIQUE DU CORTEX CEREBRAL DANS LE SYNDROME DE LA DEMENCE SENILE

par

M. COLLARD

L'incidence des arteriopathies dans divers syndromes psychiques du sujet age et plus particulierement dans la deterioration ou la confusion mentale est difficile a evaluer quantitativement.

La moitie environ des demences seniles est mixte: elles resultent a la fois de lesions degeneratives et de lesions vasculaires se caracterisant par des foyers de demyelination de la substance blanche et des ramollissements corticaux et sous corticaux.

Dans l'ischemie cerebrale d'origine tronculaire resultant d'une pathologie interessent les arteres cerebrales principales, les lesions sont engendrees par une modification de facteurs hydrodynamiques. Par contre, dans l'ischemie secondaire a des lesions interessent les arterioles corticales le processus est plus diffus et ne presente pas de localisation preferentielle dans le carrefour parieto-temporo-occipital.

Nous avons etudie 32 patients presentant un syndrome de demence senile a differents stades evolutifs et nous avons compare la clinique aux modifications arterielles mises en evidence par l'angiographie carotidienne et vertebrale in vivo et aux alterations arteriolaires et arteriolo-capillaires demontrees par la microangiographie.

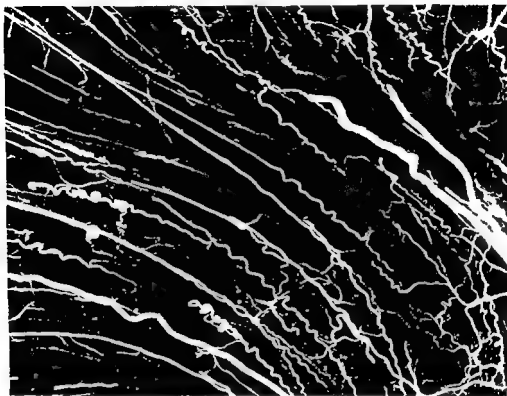


Fig 4 Microangiographie Agrandissement  $\times 100$  ■ Lésions évoluées de surcharge artérielle avec un aspect extrêmement tortueux des artérioles de premier et de second ordre Syndrome de démence sénile avec détérioration mentale grave

### Resultats

Nous n'avons retenu pour notre étude que les observations comportant des documents microangiographiques de bonne qualité avec une opacification des artérioles les plus corticales, sans artefact ni extravasation.

Les lésions d'athéromatose se caractérisent par un aspect tortueux et erpigneux des artérioles situées dans les axes des circonvolutions cérébrales. Dans les formes débutantes les artères pathologiques alternent avec des artères d'aspect normal tandis que dans les formes les plus évoluées nous observons une accentuation du processus de surcharge artérielle avec interruption de certaines artérioles. En outre sur certains documents nous observons un remplissage veineux précoce tout particulièrement dans les territoires corticaux qui ne sont plus vascularisés à la suite d'une obturation artériolaire.

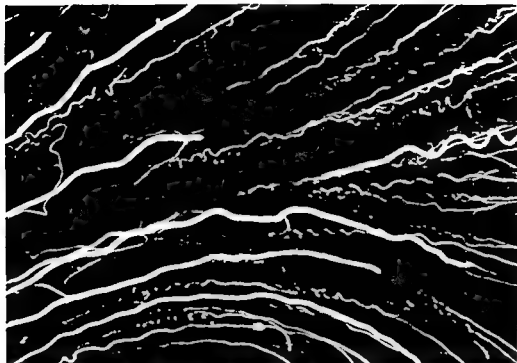


Fig 5 Microangiographie Agrandissement  $\times 22$  Atheromase très precoce malgre une injection relativement limitée du réseau arteriolaire

Lorsque nous comparons les lésions tronculaires principales mises en évidence in vivo ou au cours de l'injection post mortem aux altérations arteriolaires corticales nous constatons que les lésions arteriolaires corticales ont plus nettes chez les patients dont les artères carotides ou vertébrales conservent un aspect normal. A l'opposé les lésions arteriolaires corticales ont plus discrètes chez les patients présentant une sténose carotidienne ou vertébrale.

L'étude anatomique classique objective des lésions ischémiques qui se caractérisent par une démyélinisation de la substance blanche, un élargissement des espaces perivascularaires, une rarefaction neuronale et parfois une atrophie granulaire corticale.

### Discussion

Les formes de démence senile secondaires à des lésions dégénératives du tissu cérébral sans artériopathie, se manifestent par une diminution globale de l'intellect, des troubles mnésiques importants et enfin une détérioration mentale globale.



Fig 6 Microangiographie Agrandissement  $\times 40$  Lésions extrêmement évoluées de surcharge artériolaire avec aspect serpigneux des artéoles artérielles

Lorsqu'un facteur vasculaire intervient des signes neurologiques focaux compliquent le tableau dementiel et leur intérêt diagnostique est important mais malheureusement leur démonstration peut être difficile en raison de l'état psychique du patient.

Les traces électroencéphalographiques n'objectivent aucune localisation particulière et traduisent simplement un ralentissement plus ou moins marqué de l'électrogénèse dépendant d'une diminution de la consommation d'oxygène sans qu'il ne soit possible de l'attribuer à un processus vasculaire ou à un processus cellulaire. En outre, il n'existe aucune relation significative entre les dysrythmies lentes et la diminution de l'intellect du malade.

Dans l'ischémie cérébrale secondaire à une altération des artères carotides ou vertébrales, les lésions sont surtout localisées dans le carrefour pariéto-temporo-occipital et entraînent une ischémie relativement systématisée avec des troubles neurologiques focaux sans aggravation perceptible de la démence. Par contre, dans les observations qui se caractérisent par un aspect relativement normal des artères de premier ordre et une atteinte élective du réseau artériolo-capillaire cortical, nous avons constaté une nette aggravation du syndrome de démence.

senile sans modification neurologique focale, hormis des troubles du langage qui peuvent parfois apparaître mais qui sont très difficiles à définir chez un malade dont le comportement psychique est altéré.

### Conclusion

La microradiographie permet une estimation qualitative et quantitative des lésions artériolaires corticales puisque l'ensemble du cerveau est étudié d'une manière sérieuse par des coupes de 3 mm d'épaisseur. Les documents microangiographiques démontrent une relation significative entre la gravité du syndrome de démence sénile et le degré d'atteinte des artéroles corticales.

La détérioration mentale est la plus grave chez les patients qui présentent des lésions évoluées d'athéromasie au niveau des artéroles axiales du cortex, quelle que soit l'importance relative des lésions diffusées de ramollissement cérébral.

Les formes de démence sénile sans composante vasculaire se caractérisent par une confusion mentale et des troubles mnésiques moins spectaculaires.

Enfin, les lésions artériolaires mises en évidence par la microangiographie sont beaucoup plus discrètes chez les patients qui présentent au niveau des artères carotides et vertébrales des lésions de sténose ou de thrombose.

### RÉSUMÉ

À l'aide de la méthode microangiographique, l'auteur démontre que les lésions artériolaires corticales constituent un facteur nettement aggravant de la démence sénile en accentuant la symptomatologie clinique, quelle que soit l'importance relative des lésions dégénératives du tissu cérébral. La microangiographie permet d'étudier les lésions artériolaires du cerveau avec une notion relativement quantitative. Enfin, l'auteur a remarqué que les lésions artériolaires distales sont beaucoup plus discrètes chez les patients présentant un processus athéromateux de caractère sténotique ou obstructif au niveau des artères carotides ou vertébrales que chez les sujets dont les artères conservent une relative intégrité.

### SUMMARY

A microangiographic technique demonstrated that changes in the smaller cortical arteries aggravate the clinical state in senile dementia irrespective of the degree of degeneration of the cerebral tissue. This technique permits the changes to be investigated and their relative number to be estimated. Lesions in the smaller arterial branches in atheromatous stenosis of the carotid and vertebral arteries occurred less frequently than when the main arteries were more or less intact.

## ZUSAMMENFASSUNG

Mit Hilfe der Mikroangiographie konnte demonstriert werden dass Veränderungen in den kortikalen Arteriolen die senile Demenz klinisch beträchtlich verschlimmern dies unabhängig von dem Zustand des Nervengewebes der Hirnrinde Die Angiographie erlaubt nicht nur das Studium der Veränderungen in den kortikalen Arteriolen sondern auch deren quantitative Abschätzung Es konnte festgestellt werden dass in Fällen in denen die grossen Arterien wie die Carotis oder Vertebralis atheromatose Stenosen aufweisen die Veränderungen in den kortikalen Arteriolen mehr isoliert auftraten als in den Fällen wo die Hauptarterien relativ frei waren

## ROTATIONAL MULTIPLE SEQUENCE ROENTGENOGRAPHY OF INTRACRANIAL ANEURYSMS

by

G CORNELIS A BELLET J VAN LAGEN PH ROISIN and E LIBON

Neuroradiology should produce the maximum information on the location, relations and morphology of an intracranial aneurysm. Anteroposterior, lateral, and oblique views must be obtained although they extend the duration of the examination and augment its danger. An investigation to obviate this was therefore undertaken into the advantages of a craniograph that was available. It must be emphasized that this communication constitutes merely a preliminary note on the essentially technical factors of the examination. The apparatus consisted of (1) a 70 mm camera able to take 6 films per second (2) an amplifier coupled to a TV chain and (3) a roentgen tube that turns with the camera in all directions around a central point under telecontrol with a midpoint of rotation corresponding to the tomographic plane zero.

The probable site of the aneurysm is carefully centred by fluoroscopy to ensure that it lies in the middle of the screen. The camera assembly is then rotated at high speed through 90° or 180° at 4 films/s and 12 ml contrast medium are slowly injected. Twenty films in all are usually obtained, a difference of angle of about 5° between each making them suitable for stereoscopy.

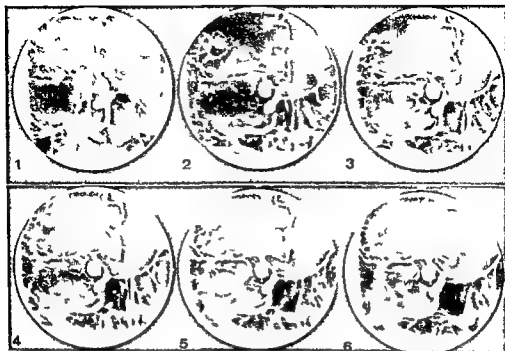


Fig 1 Early film (1) The aneurysm is not clearly defined The tube is rotated 5° between each of the following stereoscopic films (2—6) The aneurysm is progressively demonstrated

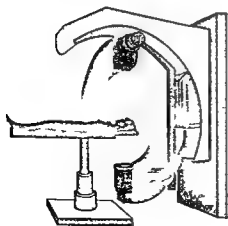


Fig 2 The apparatus



## SUMMARY

A method of examining intracranial aneurysms by rotational multiple sequence roentgenography is described

## ZUSAMMENFASSUNG

Es wird eine Methode intrakranielle Aneurysmen durch rotierende Serien Röntgenfilm Bilder zu untersuchen, beschrieben.

## RÉSUMÉ

Description d'une méthode d'examen des anévrysmes intracrâniens par angiographie en série pendant la rotation de l'appareil

## SPONTANEOUS THROMBOSIS OF VASCULAR MALFORMATIONS OF THE BRAIN

by

J I EISENMAN A ALEKOUNBIDES and H PRIBRAM

RUSSELL et coll (1963) classify cerebral vascular malformations of the brain as vascular hamartomas. The type of hamartoma includes (1) capillary telangiectasis (2) cavernous angiomas and (3) venous and arteriovenous angiomas. The term angioma does not imply a true neoplasm.

The incidence is uncertain. In the cooperative study of intracranial aneurysms and subarachnoid hemorrhage, malformations constituted 7.7% of 6,368 cases of subarachnoid hemorrhage (PERRET et coll 1966). Neurosurgical clinics publish figures as high as 4% of verified intracranial tumors (OLIVECRONA et coll 1968). COURVILLE (1963) found 22 small malformations in two years.

The natural history of vascular malformations has been recorded in many large series. The ratio of aneurysms to vascular malformations varies from 10/1 to 3/1. A number of cases of concurrent aneurysm and malformation have been reported since the advent of angiography (SALIBI 1969).

Spontaneous regression or thrombosis of cerebral malformations documented by angiography has received little attention. A survey of the literature disclosed 32 examples of this occurrence in 17 reports dealing with cerebral malformations. Only occasional reference to this finding has been made in the radiologic literature (PORTER et coll 1969). We are reporting the spontaneous thrombosis of three vascular malformations. The unique findings in one of the cases suggests a familial incidence of malformations that thrombosed.

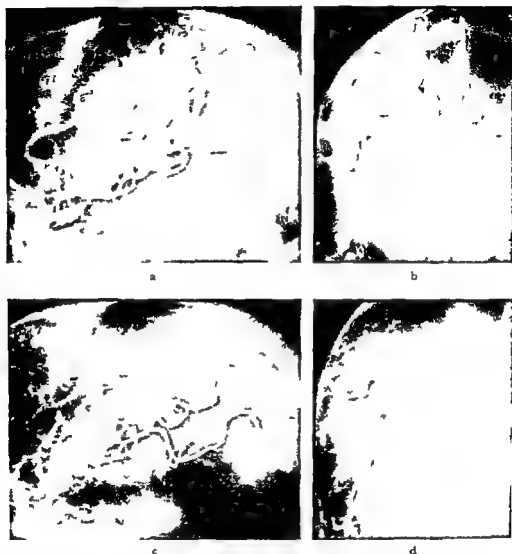


Fig. 1 a and b) Lateral and frontal views. An arteriovenous malformation in the right parietal region is well delineated. The enlarged draining vein empties into the internal cerebral vein. c and d) Four years later. No evidence of the malformation. Apparent displacement of the pericallosal artery is due to slight rotation of the head to the right.

### Case reports

*Case 1* A 34-year-old female was admitted to hospital for the third time in three years with a chief complaint of headache and progressive left-sided weakness. A fever was present. Twelve years earlier the patient had a three-hour episode of left arm numbness, weakness,

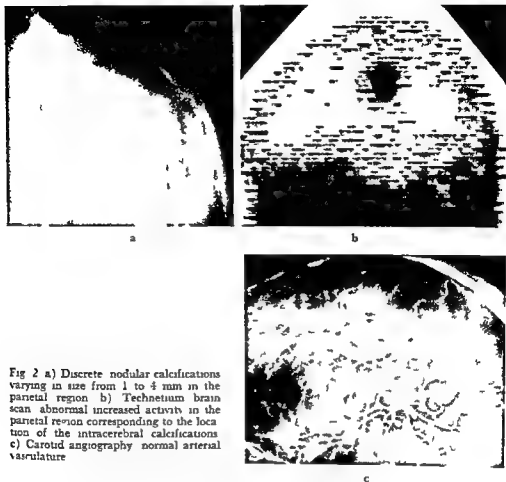


Fig 2 a) Discrete nodular calcifications varying in size from 1 to 4 mm in the parietal region b) Technetium brain scan abnormal increased activity in the parietal region corresponding to the location of the intracerebral calcifications c) Carotid angiography normal arterial vasculature

involving the left half of her body and headache. This was not treated. She had a history of occasional headaches. Eight years after this episode the patient was admitted to hospital for the first time because of progressive weakness of the left side of her body. She was unconscious. Extensor plantar responses were present bilaterally. Lumbar puncture showed an opening pressure of 600 mm water. The cerebrospinal fluid was blood tinged and xanthochromic. There were 2900 red blood cells per cubic millimeter, five white blood cells per cubic millimeter and the cerebrospinal sugar was 80 mg %.

A right carotid angiography demonstrated an arteriovenous malformation in the parietal region (Fig 1 a b). The films from the mid and late arterial phase showed drainage from the posterior parietal region into the deep venous circulation. Displacement of the midline vessels suggested intracerebral hemorrhage. Spastic hemiparesis on discharge from the hospital.



Fig 3 a) Frontal tomogram brow up from encephalography shows a mass projecting into the roof of the left lateral ventricle. Nodular calcifications are present within the mass. b) On lateral film from serial left carotid angiography there is no evidence of vascular malformation.

The second hospital admission next year was for grand mal seizure and left hemiparesis. Two days later a premature infant was delivered spontaneously. A cerebral angiography again showed the arteriovenous malformation in the right parietal region. *Physical examination* On admission this third time the patient was comatose and responded only to deep pain. Deep tendon reflexes were hyperreactive. Lumbar puncture was normal. Bilateral carotid and vertebral angiography failed to demonstrate the arteriovenous malformation previously demonstrated (Fig 1 c, d).

The patient's mental and physical status continued to improve. The left hemiparesis and spasticity residual to her first hospital admission were still present.

*Case 2* A 36-year-old male was admitted to hospital in a stuporous state with right hemiparesis. The patient had experienced seizures since the age of five. These were characterized as general convulsions and controlled with phenobarbital. Recent numbness, weakness, and tremor were noted in the right hand.

Lumbar puncture showed slight xanthochromic fluid. Spinal protein was 64 mg%, erythrocytes 26 per cubic millimeter, sugar 50 mg%, and chlorides 134 mEq/l.

Roentgen examination of the skull, brain scan, carotid angiography, and encephalography were performed. Conventional films revealed nodular calcifications in the left parietal region (Fig 2 a). The brain scan showed a hot spot in the same location as the intracerebral calcification (Fig 2 b). The carotid angiography was normal (Fig 2 c).

At surgery the bone flap in the left parietal region was thinned to 1/3 the thickness of the surrounding bone. A yellowish area was present on the cerebral cortex. The hard firm material was dissected from the surrounding normal brain. Calcific areas were noted. The mass measured 2 cm  $\times$  3 cm  $\times$  5 cm.

Microscopic sections showed the mass to be composed of numerous vascular channels and sinusoids. There were many large thin walled sinuses separated by clusters of capillaries. Much of the intervening stroma was hyalinized and numerous foci of calcification were present. A small area of cerebral cortex adherent along one margin showed hemosiderin laden histiocytes and an occasional cluster of gutter cells. The findings were those of partially thrombosed cavernous capillary angioma.

*Case 3* A 38 year-old Mexican male was admitted to hospital with right-sided weakness and slurred speech. A right central facial paralysis was present. There was extensor plantar response on the right. The lumbar puncture revealed grossly bloody fluid. Encephalography showed a faintly calcified mass projecting into the roof of the left lateral ventricle at the junction with the anterior horn (Fig 3 a). The left carotid angiography was normal (Fig 3 b). The tests for cystocerosis were negative.

*Past history* The patient's symptoms started at age 20. At that time he had a subarachnoid hemorrhage (with loss of consciousness). Progression of symptoms over the next few days included right hemiparesis, positive Kernig's sign, bilateral paresis of the 3rd, 5th and 6th nerves, dysphagia and inability to protrude his tongue. There was progressive improvement. Conventional films were normal. Vertebral angiography was technically inadequate. A few months later the patient was chair ridden because of ataxic gait. There was a right central facial palsy and an inconstant extensor plantar response. Lumbar puncture was normal. Over the years there was little change in the patient's condition. The year before admission the patient had six seizures. This was associated with dysarthria, coarse vertical nystagmus, truncal ataxia, impaired equilibrium and generalized hyperreflexia. Lumbar puncture was normal. Bilateral carotid angiography was normal.

*Family history* The patient's 46 year old sister had died as a result of a venous malformation of the third ventricle. Her angiography failed to disclose the malformation. A Torkildsen shunt was performed. At the postmortem examination the venous malformation was found and a similar lesion disclosed in both adrenals.

One of the patient's brothers is under treatment for epilepsy. Carotid angiographic examinations were normal. The second daughter of this brother was born with a sizable venous malformation on the tip of her nose.

The history, calcified mass projecting into the ventricle and family history qualify this patient's inclusion in this series of thrombosing malformations.

## Discussion

Several explanations have been proposed to explain why angiography fails to demonstrate these malformations. RUSSELL et coll (1963) state that the offending malformation may be destroyed by spontaneous hemorrhage. DAVIDOFF (1954) recorded an instance of thrombosis of a venous malformation associated with an intracerebral clot. In some instances BECK (1954) suggested that ischemia associated with a rapidly expanding lesion, compression, thrombosis or spasm of vessels may be the cause. Arteriosclerosis is suggested by POOL et coll

Table

*Cases of malformations not demonstrated or diminished in size at angiography collected from the literature*

Reference and year	No of cases	Age	Sex	Angiographic findings
CASTAIGNE (1961)	1	31	F	Mass
CRAWFORD & RUSSELL (1956)	2	32	M	Mass
		8	M	Mass
DECKER (1966)	1	Not available		Normal
FLRTADO et coll (1951)	1	37	F	Mass
HOOK & JOHANSON (1958)	1	54	M	Malformation disappeared on following examination 21 years later
JAIN (1966)	1	15	M	Normal (intraventricular malformation)
JENSEN et coll (1963)	2	Not available		Malformation not found
KAMRIN & BLCHSBAUM (1963)	2	15	F	Mass
		46	F	Mass
KRAYENBUHL & SIEBENMANN (1965)	11	30	M	Mass
		30	M	Mass
		32	M	Poor contrast filling
		48	M	Mass
		15	F	Mass
		15	M	Mass
		20	F	Mass
		40	M	Mass
		69	M	Mass
		51	F	Mass
		41	M	Mass
KUSHNER & ALEXANDER (1970)	1	46	M	Malformation diminished in size on subsequent examinations
MOYES (1969)	1	17	M	Mass
NORLÉN (1949)	1	Not available		Malformation much smaller than surgical specimen
PAPATHIODOURU et coll (1961)	1	25	M	Malformation not demonstrated at angiography
PATERSON & McHISOCK (1956)		14	M	Normal
		27	F	Normal
SALIBI (1969)	2	50	M	Mass
		20	M	Mass
SCHNEIDER & LISS (1958)	1	31	I	Mass
SVEN & PESERICO (1958)	1	46	F	Malformation decreased in size in 35 years

(1965) as a factor influencing the spontaneous regression of malformations. The association of subarachnoid bleeding with all of the cases suggests that either the accumulated hematoma or cerebral edema compressed the malformation caused slow blood flow through the malformation and predisposed to thrombosis. BOGREN et coll (1970) suggested that poor angiographic demonstration of cavernous hemangioma may be due to the small size of the feeding arteries. The large vessels demonstrated angiographically were venous.

Of the 32 cases in the literature 28 malformations were not demonstrated at angiography. Two malformations diminished in size on repeated angiography. In one case the malformation was not demonstrated on the second examination. In one case the angiographic size was disproportionately smaller than the surgical specimen (Table).

In reviewing the problem of obscure causes of subarachnoid hemorrhage CRAWFORD et coll (1956) analysed 21 cases in which the cause of spontaneous cerebral hemorrhage was remote because of the small size or location of the offending lesion. The anatomic examination disclosed the etiology to be an arteriovenous or venous hamartoma of the brain. In view of the difficulty in diagnosis the name cryptic hamartoma was proposed to describe these lesions. McCORMICK et coll (1956) collected 260 cryptic malformations from the literature. Combined with their 48 cases the total was 308 cases. 161 cases were supratentorial in location.

GERLACH et coll (1961) referred to these cryptic hamartomas as microangiomas. They have shown that a higher proportion can be detected by serial angiography. Magnification angiography in the examination of intracerebral hematomas should offer greater prospect for identification of these microangiomas.

Failure to demonstrate a malformation at technically adequate angiography does not exclude vascular malformation from the differential diagnosis of subarachnoid hemorrhage especially if a mass is present.

Evaluation of the results of non surgical therapy such as radiation therapy is certainly to be questioned. Cures attributed to therapy could just as well have occurred spontaneously.

### Acknowledgement

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### SUMMARY

Three patients with spontaneous thrombosis of vascular malformations of the brain are presented. Thirty two such cases collected from the literature are reviewed. Patients with



subarachnoid hemorrhage should have vascular malformation considered in the differential diagnosis even when the lesion is not demonstrated by cerebral angiography

## ZUSAMMENFASSUNG

Drei Patienten mit einer spontanen Thrombose von Gefassmissbildungen des Gehirns werden vorgestellt. Zweunddreissig derartige aus der Literatur gesammelte Falle werden besprochen. Fur Patienten mit einer Subarachnoidalblutung sollte bei der Differentialdiagnose eine Gefassmissbildung erwogen werden auch wenn sich bei der cerebralen Angiographie eine derartige Lasion nicht nachweisen lasst.

## RÉSUMÉ

Présentation de deux cas de thrombose spontanée de malformation vasculaire du cerveau. Les auteurs passent en revue trente deux cas analogues recueillis dans la littérature. Il faut penser à une malformation vasculaire dans le diagnostic différentiel des hémorragies sous arachnoïdiennes même quand l'angiographie cérébrale ne montre pas la lésion.

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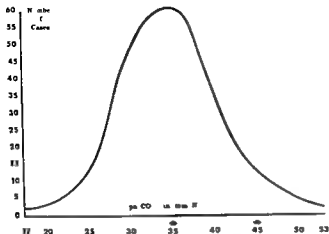


Fig 1 Number of cases versus  $\text{paCO}_2$  level. Normal range indicated by arrows

local anesthesia during which the arterial  $\text{paCO}_2$  had been determined at least once

**Method** The radiographic reports of these 735 cases were reviewed and those meeting the following criteria were evaluated (1) Direct percutaneous carotid angiography done under local anesthesia (2) Arterial  $\text{paCO}_2$  determinations available from blood drawn from the carotid needle at the time of contrast injection (3) Contrast medium visible in the carotid artery in the neck for two seconds or less (4) Films of quality satisfactory for the evaluation of late venous filling (5) No evidence of intrinsic vascular disease or mass effects

One hundred thirty cases were found whose reports fit our criteria. Careful reevaluation of the films showed minor abnormalities which led to our discarding an additional 64 cases leaving 66 hemispheres judged morphologically normal. These 66 hemispheres were not necessarily those of normal patients. They were from our routine angiographic patients and all had sufficient clinical abnormality to indicate cerebral angiography. These cases were not elected on any clinical grounds and when reviewed clinical information was purposely withheld until after the circulation times had been determined.

Our arterial  $\text{paCO}_2$  determinations were made on heparinized blood iced as soon as drawn and measured shortly thereafter. Before June 1969 the determinations were made with a Beckman Gas Analyzer Model LB-1 and afterward on an Instrumentation Laboratory PH/Gas Analyzer Model 112.

The hemisphere arteriovenous circulation time and the regional venous appearance times were determined by the method of GREITZ (1968) by three observers. Methylglucamine iothalamate (Conray 60) was the only contrast

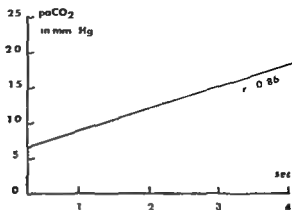


Fig 2 Change in arteriovenous circulation time versus  $\text{paCO}_2$  level

medium used, 10 ml was the dose for common carotid injections 5 ml for internal carotid injections. Mechanical injections were used routinely, but some injections by hand were also included. All had contrast in the cervical carotid artery for two seconds or less.

Nine patients with normal circulation times and normal arterial  $\text{paCO}_2$  levels had a repeat lateral film series done after hyperventilation induced hypocapnia and a repeat  $\text{paCO}_2$  determination, and three patients had a repeat lateral series during hypercapnia induced by administration of a mixture of 10%  $\text{CO}_2$  and 90% oxygen.

### Results

Using the criteria for normal described by LEEDS & TAVERAS (1969) none of our cases showed abnormal venous filling that could be attributed to an abnormal  $\text{paCO}_2$  level either in the routine cases or in the 12 cases with induced hypercapnia or hypocapnia. Each of these latter cases served as its own control. Five of the nine hypocapnic cases had  $\text{paCO}_2$  values of 20 mm or less after hyperventilation and the three patients who inhaled 10%  $\text{CO}_2$  had  $\text{paCO}_2$  elevations of 12 mm, 13 mm and 7 mm respectively, to raise their arterial  $\text{pCO}_2$  values above 45 mm Hg.

Fig 1 shows the distribution of the 735 arterial  $\text{paCO}_2$  values with the range usually considered normal in the literature indicated by the arrows. The mode of the distribution curve is at a lower value than the standard normal value of 40 mm Hg. Our usual  $\text{paCO}_2$  range at angiography, containing 90% of our determinations is 28 to 45 mm mercury. We speculate that our slightly lower range of  $\text{paCO}_2$  values reflect mild hyperventilation due to apprehension in the unanesthetized patient.

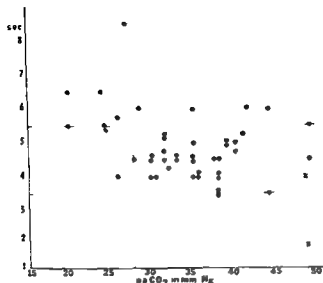


Fig 3 Arteriovenous circulation time versus  $\text{paCO}_2$  level

Fig 2 demonstrates the changes seen in the small series of artificially varied  $\text{paCO}$  levels. Nine cases had decreased  $\text{paCO}$  levels after hyperventilation and three cases had increased  $\text{paCO}$  levels after  $\text{CO}$  inhalation. A strictly linear relationship was not found, but reduction in  $\text{paCO}$  uniformly causes an increase in circulation time and vice versa. The ordinate represents millimeters of change in arterial  $\text{paCO}$  values induced by manipulations. The line shown is a line of best fit for the data obtained by the method of least squares and shows good inverse correlation between  $\text{paCO}$  level and arteriovenous circulation time. Our data indicate an average change of 3.5% in the arteriovenous circulation time per millimeter of change in  $\text{paCO}$ ; the range being 2.6 to 5.4%. This series is presently too small to be clinically useful.

Fig 3 shows the circulation times of our 66 morphologic normals plotted against their carotid artery  $\text{paCO}$  levels. As noted above, these cases were chosen only because they had no recognizable angiographic morphologic abnormality; they were clinically abnormal enough to be referred for angiography. We elected 3.5 to 5.5 seconds as our normal range for arteriovenous circulation time while realizing that some investigators would have placed the normal range for circulation time somewhat lower than this.

The central box of Fig 3 contains those hemispheres with normal  $\text{paCO}$  levels and normal hemisphere arteriovenous circulation times. The box on the upper left contains those hemispheres with low  $\text{paCO}$  levels and prolonged

hemisphere arteriovenous circulation time Although no patients in our series had naturally occurring high  $\text{paCO}_2$  levels, two normal cases developed abnormally short circulation times when given 10 %  $\text{CO}_2$  by mouthpieces These are marked  $\lambda$  The third  $\lambda$  is a case whose circulation time was changed from prolonged to normal by inducing hypercapnia

The center left box in Fig 3 contains 4 hemispheres with low  $\text{paCO}_2$  levels and normal circulation times but three of the normal circulation times were borderline on the high side A small bubble of air left in the sampling syringe may cause a falsely low  $\text{paCO}_2$  value this is postulated as an explanation for the other cases although this cannot be proved in this retrospective investigation

The upper center and upper right boxes in Fig 3 show seven hemispheres with normal or high  $\text{paCO}_2$  levels and prolonged arteriovenous circulation times which is the reverse of the usual response It is postulated that this could be the only angiographic manifestation of abnormality in an abnormal hemisphere The clinical records of the seven patients were reviewed Four of the seven clinically had definite organic brain disease, but morphologically normal hemispheres Their flow patterns were clearly abnormal The remaining three hemispheres with flow abnormality were less clear clinically one had temporal lobe epilepsy one had mild generalized brain atrophy and one had no definite clinical diagnosis

## Discussion

Although morphologic information is the predominant interest of most clinicians utilizing cerebral angiography a great deal of information may be obtained by evaluating the rate of transit of the contrast medium through the cerebral vessels Most published work on regional alterations in transit rate stresses the utilization of rapid or early venous filling to diagnose such entities as tumor, arteriovenous malformation or areas of necrosis with loss of autoregulation of the arteriolar capillary bed at the periphery of the damage area (LEEDS & TAVERAS TAVERAS et coll ) Regional or focal low venous filling may be seen in areas of edema or contusion (DAVIS & COXE) Considerable information may be obtained by evaluating the overall arteriovenous circulation time as well This is often slowed in patients with increased intracranial pressure or generalized brain edema and less often and less reliably in patients who have generalized increased vascular resistance secondary to atherosclerotic cerebral vascular disease

While this latter information may not be as useful to the clinician since it is not a guide to definitive therapy it is of importance in furthering the

understanding of the effect of these diseases on brain flow. Thus in those cases where there is generalized slowing of the angiographic arteriovenous circulation time, it is important that the effect of the  $\text{paCO}_2$  on the circulation time is appreciated so that false positive interpretations are not made. In addition the demonstration of arteriovenous shunts by any type of angiography may be enhanced by manipulation of the arterial  $\text{paCO}_2$ . If as is usually the case the vessels of the shunt do not retain their  $\text{CO}_2$  reactivity while the remaining brain vessels do, hyperventilation to induce generalized hypocapnia will increase the resistance of the normal vessels and relatively increase the amount of flow through the nonreactive shunt region. Finally there is a theoretical possibility that multiple injections of contrast medium into hypocapnic vessels resulting in increased contact of the contrast medium with the blood brain barrier due to the slowed flow may cause damage to this barrier and cause an increase in the complications of angiography. In other words this may induce generalized brain edema secondary to the contrast medium much as was occasionally seen with the less optimal contrast media previously used for cerebral angiography. In view of these considerations we feel that it is important to once again call attention to the effect of the blood  $\text{CO}_2$  levels on the brain vessels.

Since there was no increase in the complication rate of our angiographies done under local anesthesia we feel justified in concluding that the premed slight hyperventilation in patients with local anesthesia does not adversely affect the blood brain barrier within this range of  $\text{paCO}_2$ . We also feel justified in continuing to use the limits of usual range of  $\text{paCO}_2$  levels in evaluation of arteriovenous circulation times using 3.5 to 5.5 seconds as the normal time limits. Granted that we were unable to evaluate clinically asymptomatic normal individuals most of our patients with morphologically normal hemispheres and without clinical evidence for decreased flow had normal arteriovenous circulation times within the arterial  $\text{paCO}_2$  values encompassed in our normal range. Suggesting this was the finding that a reduction of  $\text{paCO}_2$  below this range consistently prolonged the circulation time and elevation of the  $\text{paCO}_2$  above this normal range reduced the circulation time of the three patients who inhaled 10%  $\text{CO}_2$ . Of passing interest is the finding that experimental changes in arterial  $\text{paCO}_2$  in 12 cases failed to produce any variation in the normal filling sequence. This certainly could stand the test of an increased number of cases.

### Acknowledgement

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## SUMMARY

The cerebral angiographic circulation times of 66 normal cases were correlated with  $\text{paCO}_2$  levels during angiography. Nine patients then were made hypocapnic, three hypercapnic, and the angiographies and  $\text{paCO}_2$  levels were repeated. No abnormal venous filling sequence was thus induced. Seven of the 66 cases had abnormal arteriovenous circulation times as the sole manifestation of their clinical abnormality. Diagnosing an abnormal hemisphere due to prolonged arteriovenous circulation time alone is invalid without a concomitant  $\text{paCO}_2$  determination.

## ZUSAMMENFASSUNG

Die cerebralen angiographischen Zirkulationszeiten von 66 normalen Fällen wurden zu den  $\text{paCO}_2$  Spiegeln während der Angiographie korreliert. Neun Patienten wurden anschliessend hypokapnisch und drei hyperkapnisch gemacht und die Angiographie und Bestimmungen des  $\text{paCO}_2$  Spiegels wiederholt. Es wurde somit keine abnorme venöse Füllungssequenz hervorgerufen. Sieben der 66 Fälle hatten eine abnormale arteriovenöse Zirkulationszeit als einzige Manifestation ihrer klinischen Abnormalität. Eine abnormale Hemisphäre alleine aus einer verlängerten arteriovenösen Zirkulationszeit ohne gleichzeitige  $\text{paCO}_2$  Bestimmungen herzuleiten ist unzureichend.

## RÉSUMÉ

Les temps de circulation cérébrale angiographique dans 66 cas normaux ont été confrontés avec les valeurs de  $\text{paCO}_2$  au cours de l'angiographie. Puis neuf patients ont été rendus hypocapniques, trois hypercapniques et les angiographies et les mesures de  $\text{paCO}_2$  ont été répétées. Ces modifications de  $\text{paCO}_2$  n'ont pas entraîné de modifications anormales du moment d'opacification des veines. Sept de ces 66 cas avaient des temps de circulation artérioveineuse anormaux qui étaient la seule manifestation de leur anomalie clinique. L'allongement du temps de circulation artérioveineuse ne permet pas de faire le diagnostic d'hémisphère cérébral anormal sans une mesure concomitante de la  $\text{paCO}_2$ .

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## ERGEBNISSE DER SCHICHT ANGIOGRAPHIE DER HINTEREN SCHADELGRUBE

von

P. GERHARDT UND P. OLDENKOTT

Mit der technischen Verbesserung der Röntgengeräte haben wir die Möglichkeit die diagnostische Aussage von Spezialuntersuchungen wesentlich zu verbessern. Dies ist zum Beispiel für die Neuro Radiologie der Fall bei der Anwendung der Vertebralstomographie, wie wir sie seit etwa 1 1/2 Jahren in Tübingen durchführen. Der Vorteil dieses Untersuchungsverfahrens als Ergänzung zur Übersichts-Angiographie der hinteren Schädelgrube liegt in der Überlagerungsfreien Abbildung einzelner Gefäßabschnitte.

Die Röntgenaufnahmen werden mit dem Mimer Universal der Firma Flema Schöninger angefertigt. Als Zusatzgerät dient ein fahrbares Bildverstärkerstativ Biwop III mit einem Siemens-Bildverstärker Sirecon 17. Im Anschluß an die Übersichts-Angiographie erfolgt die simultane Tomographie mit fünf Schichtaufnahmen bei einem Abstand von je 0,5 cm.

Das Kontrastmittel wird durch einen transfemorale in die linke Arteria vertebralis eingeführten Katheter injiziert. Der Vorteil der Katheter-Darstellung liegt in der guten Umlagerungsmöglichkeit des Patienten ohne daß, wie bei der perkutanen Punktion, die Nadel aus dem Gefäßlumen herausgleiten kann. Bei jedem der drei Untersuchungsabläufe (a) p. Übersichtsaufnahmen, seitliche Übersichtsaufnahmen und c) tliche Schichtaufnahmen) werden 10 bis 12 ml Kontrast

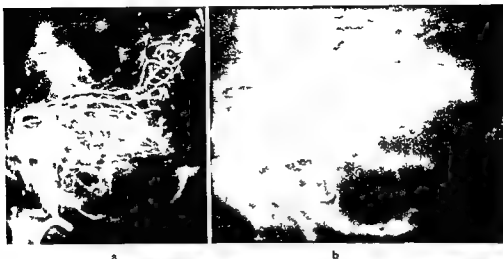


Abb 1 a) Seitliche Übersichts Angiographie einer Patientin auf der ein pathologischer Befund nicht mit Sicherheit zu erkennen ist b) Die seitliche Tomographie ergibt ein harmonische Rundung der Gefäßäste der Arteria cerebelli inferior posterior bei gleichzeitiger Stauchung derselben und geringer Ventralverlagerung der Aufzweigung in die beiden Hauptäste. Operativ wurde bei dieser 23-jährigen Patientin eine taubeneisgroße Geschwulst im Bereich der rechten Kleinhirn Hemisphäre nachgewiesen

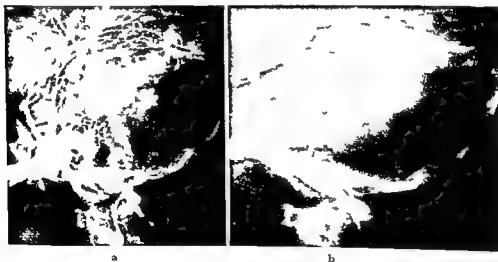


Abb 2 a) Das seitliche Vertebrales Angiogramm dieses 8-jährigen Jungen zeigt eine deutliche Verlagerung der Arteria basilaris nach ventral und der Arteria cerebelli inferior posterior nach caudal. Die Äste der Arteria cerebri posterior verlaufen konvexbögig nach cranial ausgespannt und man sieht hier pathologische Gefäße. b) Das seitliche Tomogramm 1,5 cm links paramedian ergibt einen überlagerungsfreien Verlauf der eben beschriebenen Gefäße wobei insbesondere pathologische Gefäße im Bereich der Arteria cerebri posterior und der Arteria cerebelli inferior posterior zu erkennen sind

mittel injiziert so daß eine Gesamtmenge von 30 bis 36 ml Kontrastmittel verwandt werden

Um eine Hypoxämie zu vermeiden wird der Katheter jeweils nach der Injektion aus der Arteria vertebralis zurückgezogen und vor der nächsten Injektion unter Durchleuchtung Kontrolle in das Gefäß eingeführt

Wir haben bisher 36 Patienten mit diesem Verfahren untersucht. Bei Kindern, bei Patienten mit Arteriosklerose die die selektive Angiographie nicht zuläßt und bei Notfall Angiographien wurde weiterhin die perkutane Punktion der Arteria vertebralis ohne Tomographie durchgeführt

Die bisherigen Erfahrungen zeigen daß vornehmlich der Verlauf der Arteria cerebelli inferior posterior durch die Schichtuntersuchung besser beurteilt werden kann

Die beiden in den Abbildungen gezeigten Fälle sind Beispiele dafür, daß die Tomographie den Nachteil der Überlagerung bei den Übersicht Angiogrammen aufhebt. Unsere Erfahrungen bestätigen bei annähernd allen untersuchten Patienten daß dieses Verfahren für die Routine Diagnostik eine brauchbare Ergänzung zur Übersicht Angiographie darstellt

## ZUSAMMENFASSUNG

Es wird über eine Untersuchungsmethode berichtet die es erlaubt einzelne Gefäße der Arteria vertebralis überlagerungsfrei zu beurteilen. Die Angio Tomographie der Gefäße der hinteren Schädelgrube erfordert einen etwas größeren zeitlichen Aufwand als die Übersichts-Angiographie und ist vorwiegend nur dann durchführbar wenn die transfemorale Katheter Untersuchung bevorzugt wird. Die hier berichteten Untersuchungen wurden mit dem Mimer Universal angefertigt. Es liegen Erfahrungen bei 36 Patienten vor. Diese Erfahrungen berechtigen zu der Annahme daß dieses Untersuchungsverfahren breiteren Raum in der Routinediagnostik gewinnen wird.

## SUMMARY

A method of demonstrating the branches of the vertebral artery without undue superimposition is described. Admittedly, tomangiography of the vessels of the posterior fossa is more time consuming than ordinary angiography and is practicable only after a transfemoral approach. A total of 36 investigations were performed with the aid of the Mimer. The authors feel that their method yields superior results and will become a standard procedure.

## RÉSUMÉ

Les auteurs décrivent une technique d'examen qui permet d'étudier des branches de l'artère vertébrale sans superposition. L'angiographie des vaisseaux de la fosse postérieure prend un peu plus de temps que l'angiographie simple et n'est réalisable que quand l'injection est faite par cathétérisme de l'artère fémorale. Les examens présentés ont été faits avec le Mimer Universal sur 36 malades. Les résultats permettent de penser que cette technique d'examen occupera une place plus importante dans le radio diagnostic courant.

## CEREBRAL ANGIOGRAPHY DURING A MODIFIED VALSALVA MANOEUVRE UNDER GENERAL ANAESTHESIA

by

T D HAWKINS and D POWELL

The influence of low arterial  $p\text{CO}_2$  tensions induced by hyperventilation under general anaesthesia on the angiographic appearance of intracranial tumours has been reported previously (GRANGE et coll 1969). Under these conditions there is slowing of the cerebral circulation, reduction in the calibre of normal cerebral arteries and veins and an improvement in the demonstration of tumour vessels of both gliomas and meningiomas in most cases.

It has been suggested that this technique might be hazardous and that slowing of the cerebral circulation by other means might be safer and equally informative diagnostically.

One method of slowing the cerebral circulation is to reduce the cardiac output which can be achieved by elevation of the intrabronchial pressure either by the Valsalva manoeuvre or by inflation of the lungs.

The classical Valsalva manoeuvre, forced expiration against a closed glottis, produces a rise in intrathoracic pressure. This causes the cardiac output to fall due to diminished venous return and a consequent fall in arterial systolic and

pulse pressures and a rise in central and peripheral venous pressures. In the normal conscious patient the baroreceptor reflex compensates for the reduction in cardiac output and on release of the intrathoracic pressure there is a characteristic overshoot in arterial pressure with a secondary bradycardia. In the anaesthetised patient, the baroreceptor reflex is partially or completely blocked and a compensatory overshoot of arterial pressure does not occur (WYLIE & CHURCHILL DAVIDSON 1966; SCOTT *et coll.* 1969).

The use of the Valsalva manoeuvre in contrast examinations of the cardiovascular system in man has been described by CELIS *et coll.* (1956). The success of this technique however is dependent on the active co-operation of the patient. Inflation of the lungs to a predetermined pressure in a sedated or anaesthetised subject is a more certain method of producing the required effect on the circulation. BOEREMA & BLICKMAN (1955) have shown in anaesthetised dogs that elevation of the intrabronchial pressure to between 40 and 60 cm water causes compression of the right heart and venae cavae which leads to a fall in cardiac output and systolic arterial pressure. These authors were the first to demonstrate experimentally the diagnostic possibilities of this method in cardioangiography.

The experimental and clinical aspects of the intrabronchial pressure technique were further elaborated by NORDENSTROM (1960, 1963) who employed this method when performing cardiovascular pulmonary and renal angiographic studies in man.

A similar modified Valsalva manoeuvre under general anaesthesia has been used by ZACIS *et coll.* (1964) to achieve cerebral panangiography by injection of contrast medium into one carotid artery.

In the present investigation of 25 patients suffering from a subarachnoid haemorrhage or intracranial tumour cerebral angiography was performed under general anaesthesia during a modified Valsalva manoeuvre to establish whether the method is safe and to observe the effect on the cerebral circulation.

The angiograms were compared with those obtained on the same patient during spontaneous respiration. In suitable cases the method was also compared with the hyperventilation technique.

**Technique.** All the patients had a general anaesthetic. They were induced with thiopentone and were intubated with a cuffed tube following a dose of suxamethonium chloride. Spontaneous respiration was allowed to return. Anaesthesia was maintained with nitrous oxide (6 litres) and oxygen (3 litres) supplemented by trichlorethylene.

Angiography was performed by direct puncture of the common carotid artery. One rapid film series was taken in the lateral plane. A second film series was then taken after raising the intrathoracic pressure to 40 cm of water for 5 to 6

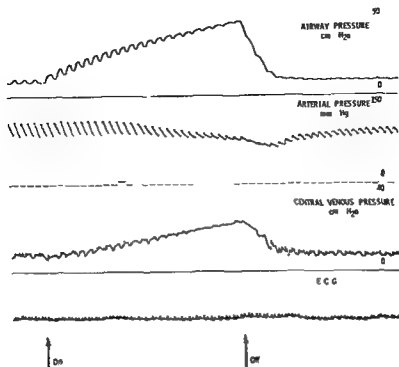


Fig 1 Valsalva manoeuvre. Typical simultaneous recording of cardiovascular responses to a raised airway pressure in an anaesthetised female aged 58 years with subarachnoid haemorrhage. This shows the rise in central venous pressure and corresponding fall in arterial pressure without significant ECG change. The arrows on the bottom of the trace indicate the beginning and end of the period of raised intrathoracic pressure.

seconds before injection of the contrast medium. The pressure was maintained at this level throughout the film series and then immediately released. The intrathoracic pressure was monitored with a spring gauge and in some the airway pressure was also measured via a side tapping in the airway. The central venous pressure was measured via a catheter in the superior vena cava and the arterial pressure via a needle in the contralateral common carotid artery.

A rise in the airway pressure was always followed within 4 seconds by a fall in the arterial pressure and a rise in the central venous pressure which returned to normal soon after the airway pressure was released. No significant ECG changes were noted (Fig 1).

In some patients an additional film series was obtained during hyperventilation the technical details of which have been described previously (SAMUEL *et coll* 1968).



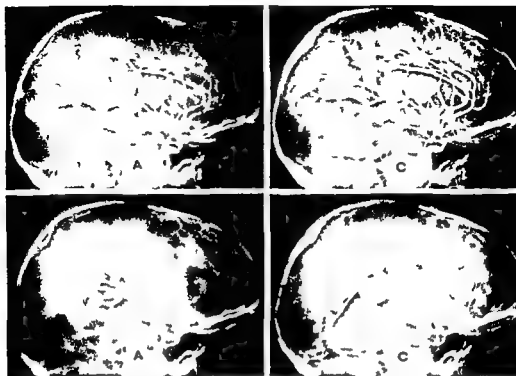


Fig 2 Case 1 Right parietal arteriovenous malformation. Rapid serial angiograms taken during spontaneous respiration (A) and a modified Valsalva manoeuvre (C). The improved demonstration of the lesion in series C is due to enhanced density of the contrast medium following non selective slowing of the cerebral circulation.

## Results

No patient appeared to suffer any ill effects from the procedure.

The response of the cerebral circulation to the modified Valsalva manoeuvre was variable. The least angiographic changes occurred in the 6 patients who had the least cardiovascular response to the manoeuvre.

The cerebral circulation was slowed in most patients.

In some the circulation time was so prolonged that a good venous phase was not obtained up to 12 seconds after commencement of the film series. This was particularly noticeable in patients with clinical evidence of raised intracranial pressure.

There was slight reduction in calibre of the cerebral arteries in a few patients, and when this occurred it was usually associated with narrowing of the extracranial internal carotid artery.

There was some improvement in the density of the contrast medium in the

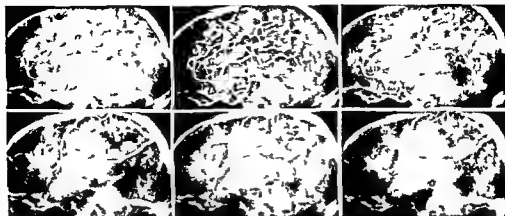


Fig 3 Case 2 Left temporal glioma. Spontaneous respiration (A) low arterial  $pCO_2$  induced by hyperventilation (B) and modified Valsalva manoeuvre (C). Tumour vessels are seen in the anterior temporal region in all three series (upper row). Early venous drainage into the basal vein in series B only (arrows). In the venous phase (lower row) the basal and temporal veins appear much the same in all three series but their significance is more apparent in series B (arrows) when the normal cortical veins are reduced in calibre.

cerebral vessels in the majority of patients. This improvement was most marked in those in whom there appeared to be dilution of the contrast medium during angiography with spontaneous respiration presumably due to a rise in  $pCO_2$  a naturally high cerebral blood flow or the anaesthetic technique.

The improved demonstration of tumour vessels produced by hyperventilation with shunting of contrast medium through the tumour — the angiographic equivalent of the inverse steal syndrome of LASSEN & PALVOLGII (1968) did not occur with the modified Valsalva manoeuvre.

In the accompanying figures those marked A are from the film series taken during spontaneous respiration and those marked B and C during hyperventilation (low arterial  $pCO_2$ ) and a modified Valsalva manoeuvre respectively.

### Case reports

**Case 1** A female aged 17 years with a right parietal arteriovenous malformation. The improvement in the demonstration of the lesion with the Valsalva manoeuvre (Fig 2C) is due to some non selective improvement in the quality of the angiogram compared with the films obtained during spontaneous respiration (Fig 2A).

**Case 2** A male aged 50 years with raised intracranial pressure due to a left temporal tumour. Tumour vessels are seen in the anterior temporal region on all three series (Fig 3). In the late arterial phase there is early filling of the basal vein in series B (low  $pCO_2$ ) which did not occur in either of the other two series. In the venous phase the left basal and temporal veins draining the tumour appear much the same in all three series but their

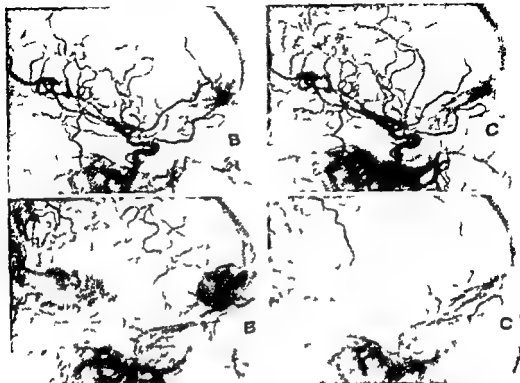


Fig. 4. Case 3. Subfrontal meningioma. Hyperventilation technique (low arterial  $pCO_2$ ) (B) and modified Valsalva manoeuvre (C). The demonstration of the tumour circulation particularly in the late arterial phase (lower row) is much improved in the series taken with a low arterial  $pCO_2$ .

significance is more apparent in series II where the normal cortical veins only are reduced in calibre. The angiographic diagnosis of a glioma rather than a meningioma was confirmed at operation.

*Case 3.* A female, aged 53 years, with severe frontal headaches, vomiting, bilateral papilloedema and frontal lobe symptoms and signs. The hyperventilation technique is compared in Fig. 4 with the Valsalva manoeuvre in the demonstration of a subfrontal meningioma. Series B was taken at a low arterial  $pCO_2$  and series C with the Valsalva manoeuvre. The superiority of the low  $pCO_2$  technique in demonstrating the tumour circulation is self-evident.

### Conclusions

Cerebral angiography performed during a modified Valsalva manoeuvre under general anaesthesia appeared to be a safe technique for the investigation of the patients suffering from a subarachnoid haemorrhage or intracranial tumour.

The effect of this manoeuvre in a majority of the patients was a slowing of the cerebral circulation and a non selective improvement of the angiograms

Although it is an alternative method to the hyperventilation technique produced satisfactory results in the investigation of patients with cerebral haemorrhage it was inferior to angiography performed under spontaneous hypocarbia in the investigation of those patients with intracranial tumours

## SUMMARY

In an angiographic study of 25 patients suffering from a subarachnoid haemorrhage or intracranial tumour the effect on the cerebral circulation of a modified Valsalva manoeuvre under general anaesthesia was compared with that of spontaneous respiration and suitable cases of hyperventilation. The technique appeared to be safe, slowed the cerebral circulation and produced a non selective improvement of the angiogram in most cases. It was inferior to the hyperventilation method for the demonstration of intracranial tumours.

## ZUSAMMENFASSUNG

Bei einer angiographischen Untersuchung von 25 Patienten mit einer subarachnoiden Blutung oder einem intracranialen Tumor wurde unter Vollnarkose auf die Wirkung der Valsalva-Manöver und in geeigneten Fällen mit der von Hyperventilation verglichen. Das Verfahren erscheint sicher, setzt die cerebrale Zirkulation herab und führt zu einer nicht selektiven Verbesserung des Angiogramms bei den meisten Patienten. Es ist der Hyperventilationsmethode zur Darstellung intracranialer Tumore unterlegen.

## RÉSUMÉ

Au cours d'une étude angiographique de 25 malades atteints d'hémorragie sous-arachnoïdienne ou de tumeur intracrânienne les auteurs ont comparé l'effet sur la circulation cérébrale d'une manœuvre de Valsalva modifiée sous anesthésie générale à l'effet de la respiration spontanée et dans certains cas de l'hyperventilation. Cette technique paraît sûre, elle ralentit la circulation cérébrale et produit une amélioration non sélective de l'angiographie chez la plupart des malades. Elle est inférieure à la méthode d'hyperventilation pour la mise en évidence des tumeurs intracrâniennes.

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## CORTICAL VEIN THROMBOSIS IN THE DOG WITH A REVIEW OF ASEPTIC INTRACRANIAL VENOUS THROMBOSIS IN MAN

by

E RALPH HEINZ DAVID GEETER and TRIGVE O GABRIELSEN

The earliest reference to intracranial venous thrombosis is that of RIBES in 1823 in which there was thrombosis of the superior longitudinal and the lateral sinuses. GOWERS in 1888 described aseptic intracranial venous thrombosis in cachectic patients. Dural sinus thrombosis has been recently reviewed by GREITZ & LIVA (1966). They gave a summary of the causes or states related to primary sinus thrombosis including the puerperium (MARTIN & SHEEHAN 1941), trauma (HOLMES & SARGENT 1915), intracranial arterial thrombosis (BARNETT & HYLAND 1953) and severe acute right ventricular failure (BARNETT & HYLAND). It could be seen after operations on the lungs (BARNETT & HYLAND) with diabetes mellitus (ASKENASY et coll. 1962) and some malignant blood diseases (BARNETT & HYLAND) but also without any demonstrable associated disease (RAY & DUNBAR 1950). Thrombosis may apparently involve only cerebral veins or dural venous sinuses or both in combination (HOLMES & SARGENT, MARTIN & SHEEHAN, BARNETT & HYLAND). At least in some cases thrombosis of one cerebral vein seems to spread to involve other cerebral veins and dural sinuses.

New interest has recently arisen with the publication of a monograph by KALBAG & WOOLF (1967) in which thirty cases are reported from the clinical

and pathologic viewpoints. Some information on angiography is presented.

Our own interest was aroused in 1966 after seeing a patient with thrombosis of the superficial cerebral veins and the superior sagittal sinus. Three additional patients with spontaneous thrombosis of the superior sagittal sinus have been seen in which angiography played a large part in the diagnosis. The three patients will be reported in detail elsewhere.

Currently there is widespread interest in cerebral accidents in relation to contraception pills (DOLL & VESSEY 1968, INAMA & VESSEY 1968). The vascular complications which have been said to occur in patients on the pill are usually venous in type. As the sequence of venous filling and emptying in such cases has received little attention it was thought that an investigation of the cerebral venous circulation in the dog before and after thrombosis of one of the cortical veins might be helpful in analyzing human cerebral phlebograms.

### Method

Carotid angiographies were done on six large mongrel dogs. After a carotid angiography a parietal burr hole was placed in relation to an appropriate cortical vein on the preliminary film. Serial films were exposed at a rate of 2 films per second for 3 seconds and one per second for 8 additional seconds. Cortical venous thrombosis was induced in each animal by injecting 0.1 ml of sodium morrhuate, a sclerosing solution, into a tiny cortical vein using a no. 27 needle.

Internal carotid angiography was done by cut down selective catheterization of the internal carotid artery similar to the method outlined by DAVIS & RUMBAUGH (1967). Thorotrast was used in volumes of 1 to 2 ml. The animals were sacrificed at intervals from 1 hour to 5 days after the thrombosis. Repeat carotid injections were attempted through an indwelling Teflon catheter needle, but clotting occurred in the internal carotid artery distal to the injection site in one animal. After sacrifice of the animals the brains were irrigated *in situ* with a barium plastic gelatin mixture and after formalin fixation, high detailed roentgenograms were made. Routine histology was performed.

### Results

Four of the six animals investigated were thought to be suitable for inclusion in this investigation. In two the technical difficulties in the preparation were great, and these animals were excluded. The difficulties had mainly to do with the injection intralumenally of the sclerosing solution in the very delicate cortical veins.

Table 1  
Control animals

Dog	Maximum arterial filling to drainage last vein	Delay in last filled vein to regional venous drainage	Relation last filled vein to deep veins (vein of Galen)	Circulation time
1	7.5 s	1.0 s	0 (Empty simultaneously)	8.2 s
2	5.0 s	1.5 s	1 s before deep veins	7 s
3	5.0 s	1.0 s	3 s before deep veins	7 s
4	5.5 s	1.0 s	1 s before deep veins	8 s
Average	5.72 s	1.12 s	1.0 s before vein of Galen	7.41 s

The following results were obtained

In all cases, the occluded vein remained filled with contrast medium well after the veins immediately surrounding it (Average control 1.12 s average occlusion 3.1 s). Normally both in man and in the dog the frontal veins fill and empty first then the parietal, temporal and occipital veins. In the experimental animals there was a delay in the emptying of the obstructed vein as compared with the regional normal venous drainage of up to 3 seconds (See Tables 1 and 2).

The obstructed vein remained filled after the deep veins by an average of 3.0 seconds, in the controls the last superficial veins emptied 1.0 seconds before the deep veins.

There was a discontinuity between the demonstrated obstructed vein and superior sagittal sinus. In each case this was high up on the convexity.

There was a marked increase in the density of the contrast medium in the blocked vein probably because most blocked veins were widened (2 to 3 times control).

The appearance time of the obstructed vein was difficult to measure but there was usually some delay. Filling of the vein peripheral to the block occurred in normal sequence but the actual time of filling of the vein contiguous to the occlusion was variable.

The time from the maximal arterial filling to the end of venous drainage was slightly increased in the pathologic animals. Average delay 1.64 seconds. On the other hand the total circulation time was not increased and there was no definite regional arterial flow.



Table 2  
Cortical vein thrombosis

Dog	Dilatation of obstructed vein	Maximum arterial filling to drainage of obstructed vein	Total circulation time	Delay in obstruction over regional normal venous drainage	Obstructed vein persists after vein of Calen	Venous collateral flow	Swelling lateral view
1	3%	8.5 s	8.5 s	3.0 s	1 s	Definite	None
2	2%	7.5 s	8.5 s	2.5 s	1 s	None	None
3	2%	7.5 s	8.5 s	3.0 s	5 s	None	None
4	2% 3%	6.0 s	7.5 s	4.0 s	3 s	Definite (slight)	None
Average	2.5%	7.39 s	8.25	3.1 s	3.0 s		

*Pathology.* In each of the acceptable cases there was a demonstrated thrombosis of the cortical vein. Angiographic examination, direct visual inspection at the time of operation, and inspection after sacrifice showed no arterial injury or disruption of the surrounding cortex by artefact.

The predominant feature was the thrombosis of the cortical vein extending from 10 mm to 25 mm over the surface of the upper part of the convexity of the brain without occlusion of the superior sagittal sinus. Mild to extensive local edema, which had usually assumed its form from the craniectomy defect, was seen in all animals. On injection specimens with coronal macro sections slight concentric expansion of the cortex and subcortex with local edema was found.

### Discussion

In the last two decades considerable information about septic dural sinus thrombosis in man has been accumulated. A recent extensive review by KALBAR & WOOLF includes 30 patients, many of them with radiologic and pathologic correlations. The characteristic radiologic appearance is non-filling of the dural sinuses, usually the superior sagittal sinus, but which may include the straight and lateral sinuses. While there are a number of radiologic observations in the above monograph, and in the references of KRAJČUBIČ (1954), HUBB (1961) and GRIFITZ & LIND, there are no detailed sequential films, particularly with reference to the late arterial and intermediate and venous drainage stages. The available illustrations suggest the presence of arrested arterial perfusion either

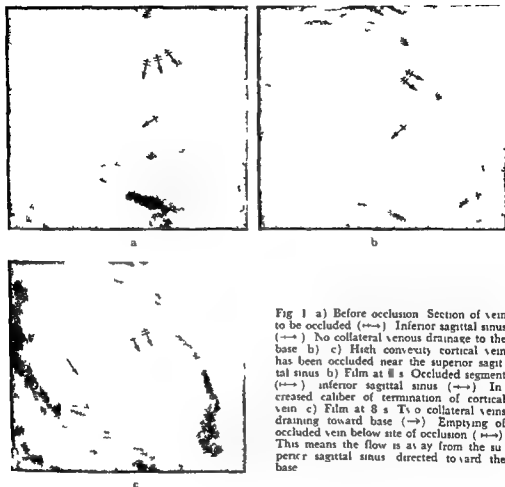


Fig 1 a) Before occlusion Section of vein to be occluded ( $\leftrightarrow$ ) Inferior sagittal sinus ( $\rightarrow$ ) No collateral venous drainage to the base b) c) High convexity cortical vein has been occluded near the superior sagittal sinus b) Film at 11 s Occluded segment ( $\leftrightarrow$ ) inferior sagittal sinus ( $\rightarrow$ ) Increased caliber of termination of cortical vein c) Film at 8 s Two collateral veins draining toward base ( $\rightarrow$ ) Emptying of occluded vein below site of occlusion ( $\leftrightarrow$ ) This means the flow is away from the superior sagittal sinus directed toward the base

generally or regionally usually with some cerebral swelling perhaps associated with out of phase filling of the internal cerebral vein or perhaps superficial collateral shunts to the transverse or cavernous sinuses

Because of the importance of making an early diagnosis in cerebral venous thrombosis we elected to induce thrombosis in a elective fashion of a cortical vein without sagittal sinus obstruction The cortical vein when thrombosed showed dramatic local dilatation and obstruction before entrance into the sagittal sinus More importantly there was an alteration in the local venous drainage in which the surrounding regional veins emptied normally or even prematurely with persistence of contrast medium in the obstructed vein and its tributaries in isolated fashion long after the normal frontal parietal temporal and occipital processes

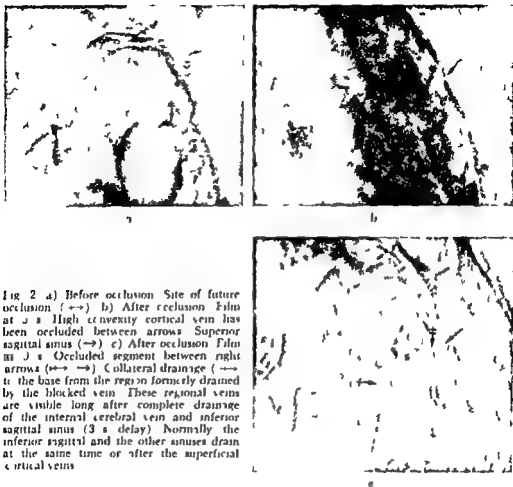


Fig. 2 a) Before occlusion. Site of future occlusion ( $\longleftrightarrow$ ) b) After occlusion. Film at 3 s. High convexity cortical vein has been occluded between arrows. Superior sagittal sinus ( $\rightarrow$ ) c) After occlusion. Film at 3 s. Occluded segment between right arrows ( $\longleftrightarrow$ ) Collateral drainage ( $\longleftrightarrow$ ) to the base from the region formerly drained by the blocked vein. These regional veins are visible long after complete drainage of the internal cerebral vein and inferior sagittal sinus (3 s delay). Normally the inferior sagittal and the other sinuses drain at the same time or after the superficial cortical veins.

sion. Indeed, there was reversal of the relationship of the normal venous drainage in that the contrast filling of the superficial convexity vein persisted from one to five seconds after deep venous emptying had occurred (Burr et coll. 1968, TAYLOR & WOOD 1961). This is a distinct reversal in regard to the normal pattern in dogs and in man.

The circulation time overall may not be lengthened because in the mildly affected subject the collateral shunts on the venous side can apparently accommodate some increased flow. Although there was no definite slowing of the arterial blood flow in our dog experiments there may be generalized as well as regional slowing of blood flow during the arterial as well as venous phases in cerebral venous thrombosis in man.

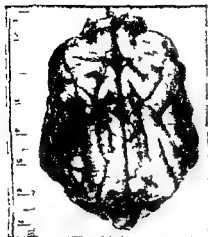


Fig 3 Top view of brain 5 hours after cortical vein thrombosis Site of occluded vein ( $\rightarrow$ ) The arteries are intact but are slightly displaced by local cerebral swelling This was confirmed by barium plastic post mortem injection The discoloration of the brain is the result of surgical trauma as well as of the venous infarct with venous congestion of the area drained by the vein

The actual site of the cortical vein occlusion could be seen in the dogs but may not necessarily be recognized in man

The increased caliber of an obstructed vein may not be readily appreciated in man because in clinical subjects a control angiogram is not usually available for comparison

Our findings in two animals indicated shunts from the mid convexity to the base resembling group 2 of KRAYENBUHL in which collateral venous channels to the base were seen (cavernous and transverse sinuses) GREITZ & LINK also demonstrated shunting to the base in two patients with aseptic superior sagittal sinus thrombosis KALBAG & WOOLF's case 32 showed collateral veins as well Our experimental results indicate that some of these veins may become available as collateral channels immediately after thrombosis of a neighboring vein

Although in the past sinography was utilized for demonstration of dural sinuses today it is not thought necessary in clinical work (KALBAG & WOOLF GREITZ & LINK) because increased volumes of contrast in aortocervical angiography will fill both carotid systems simultaneously and serve to fill the sinuses well We have used a lightly increased volume of contrast medium and cross compression of the opposite carotid artery at carotid angiography to insure adequate filling of the sinuses Obviously very late films need to be taken to see some of the delayed venous findings noted in our experimental animals

Clinically the patients with cerebral venous thrombosis are said to present classically with headache of a fluctuating character intermittent focal seizures leading to generalized seizures and finally depressed consciousness and death There is considerable evidence to suggest that many patients do not fall into

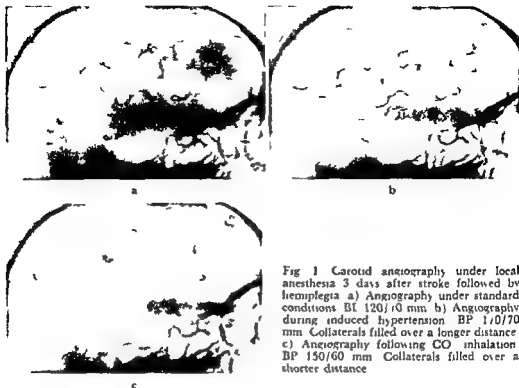


Fig 1 Carotid angiography under local anesthesia 3 days after stroke followed by hemiplegia a) Angiography under standard conditions BP 120/80 mm b) Angiography during induced hypertension BP 180/70 mm Collaterals filled over a longer distance c) Angiography following CO<sub>2</sub> inhalation BP 150/60 mm Collaterals filled over a shorter distance

A total of 27 function tests were carried out in 22 patients with angiographically demonstrated vascular occlusions

	No of tests
Rise in blood pressure	16
Hyperventilation	4
CO <sub>2</sub> inhalation	5
Papaverine	2

The blood pressure was increased with Effortil in the majority of these patients hypertensive patients were excluded from this test The number of hyperventilation examinations in this group is relatively small because most of the patients were too weak to hyperventilate satisfactorily or were aphasic and therefore unable to understand the instructions

Seventy-one function tests were performed without complications in 43 pa

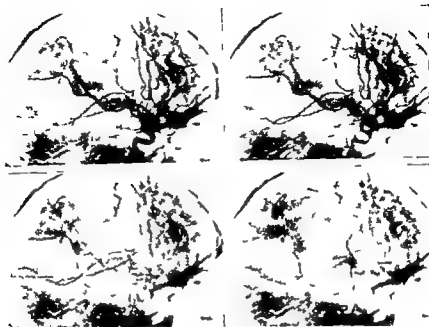


Fig 2 Malignant glioma. Angiography, under general anesthesia, BP 110/65 mm  
Lower right: Start of filling of draining vein

ients with cerebral tumours and in 5 patients with arteriovenous malformations

	No of tests
Rise in blood pressure	27
Hyperventilation	29
CO <sub>2</sub> inhalation	4
Papaverine	11

### Results

The collaterals were visible over a longer distance and were increased in number in most patients with a recent cerebral infarction and angiographically proved collateral circulation examined during pharmacologically increased blood pressure. The passage of the contrast medium through the collaterals was however usually not accelerated. This angiographic finding may consequently not correspond exactly to the subclavian steal syndrome: it means only that under increased arterial pressure the blood will be forced further into the periphery.

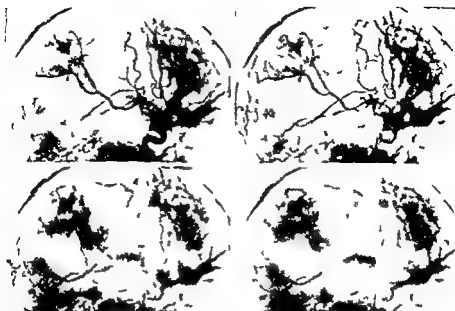


Fig 3 Same case as in fig 2. Angiographic series during induced hypertension III 170/120 mm. Upper right: Early filling of draining vein. Same speed of exposures (1 frames/s) as in fig 2.

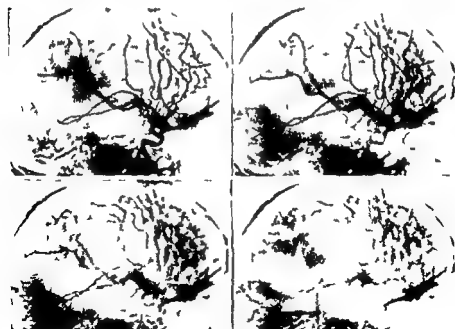


Fig 4 Same case as in fig 2. Angiographic series during  $\text{CO}_2$  inhalation. Only a few tumor vessels filled; no draining vein visible.

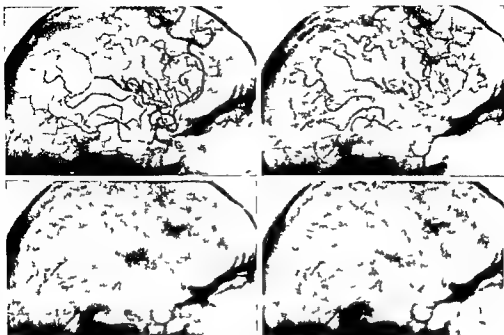


Fig 5 Astrocytoma Angiography under local anesthesia Angiographic series under standard conditions Lower left Filling of draining veins

The circulation was accelerated through the collateral region in only one patient. Acceleration with delayed antegrade circulation was evident in another patient. The raised blood pressure is obviously insufficient to overcome the increased resistance in the thrombosed vessels unless the circulation is accelerated. It could be in these patients that the raised pressure may lead to a hemorrhagic infarct, as WALTZ & SUNDT (19 20 21) have discovered in their animal experiments.

No change was evident in 3 patients and a distinctly accelerated circulation through the increased collateral circulation region occurred in one patient under hyperventilation. The collateral system in most patients was smaller in extent and visible for a shorter time after  $\text{CO}_2$  inhalation and vasodilatation with Papaverine (Fig 1).

The function test revealed no circulatory change in 4 patients, all of whom had however sustained their cerebral vascular occlusion more than four weeks before angiography.

Somewhat less than half the tumours with pathologic vessels but with no suggestion of arteriovenous anastomoses had more obvious tumour vessels under increased blood pressure but no accompanying acceleration of the circulation. Tumour blush was usually more distinct with hyperventilation especially in



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## APPLICATION OF STEREOTAXIC METHODS IN NEURORADIOLOGIC DIAGNOSIS

by

YUTAKA KURU

Stereotaxic surgery is indicated in dealing with small neural structures when a high degree of accuracy is required. Since the roentgen examination is perhaps the more important side to the technique a large number of contributions from this aspect have appeared (AMADOR et coll 1959, SCHALTENBRAND & NURNBERGER 1959, KATOH et coll 1969, TREX et coll 1969). The application of the whole method to the diagnosis appears however to have been neglected (TALLARICH et coll 1956, LICHENSTEIN 1959). An attempt has therefore been made to measure and evaluate the basal ganglia in vertebral and carotid angiograms and to apply these results in the investigation of the basal ganglia arteries as well as in the diagnosis of thalamus neoplasms.

*Materials* Two series of cases termed series A (from Osaka University Hospital) and B (from Wakayama Rosai Hospital) were examined. The magnification of objects in the series B films was measured (Fig. 1). Angiography of this series was performed by simultaneous bi plane AOT serigraphy with the tubes fixed 160 cm laterally and 120 cm anteroposteriorly respectively. For convenience distortion of the objects may be observed by superimposing a film upon the scale at a certain depth (Figs 2 c, 6 a). The exact degree of magnification of the films of the A series varied

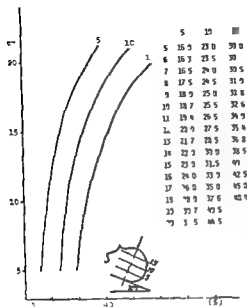


Fig 1 The graph and figures in the table indicate the magnification rate ( $M$ ) in the a.p. projection. The first column give distances from top of the film. For convenience three magnification scales are made at three different depths (cm) as an insert.

*Contribution of the method to the normal roentgen anatomy* In spite of extensive investigations of the anatomy of the basal ganglia arteries (ANDERSEN 1958 1963 WESTBERG 1963 1966, HARA & FUJINO 1966) it is always difficult to determine each perforating artery in an individual case and to name it. As for the branching, number, perforating point and course of these arteries, many variations exist. The identification of the basal ganglia arteries in an examination is considered to depend upon one or more of the following criteria: (1) in accordance with a previously determined classification; (2) by measuring the area which the artery supplies; (3) by determining the perforating point of the artery; and (4) according to the course described by the artery.

The stereotaxic method is applied to criteria (2) and (3). The usual stereotaxic reference points are marked on the encephalogram. A few local landmarks in angiograms may be used as well. These landmarks may determine the section supplied by a perforating artery and the perforating point of the artery may also be indicated. Distances in an a.p. angiogram usually provide information as to the structures present. For example, reference to the plates of the atlas by SCHALTENBRAND & BAILEY (1959) made it possible to evaluate the structure the artery in question supplied.

The film of the arterial phase in the a.p. direction is superimposed for example upon the 10 cm depth scale (Figs 1, 2 c) and the distance between the

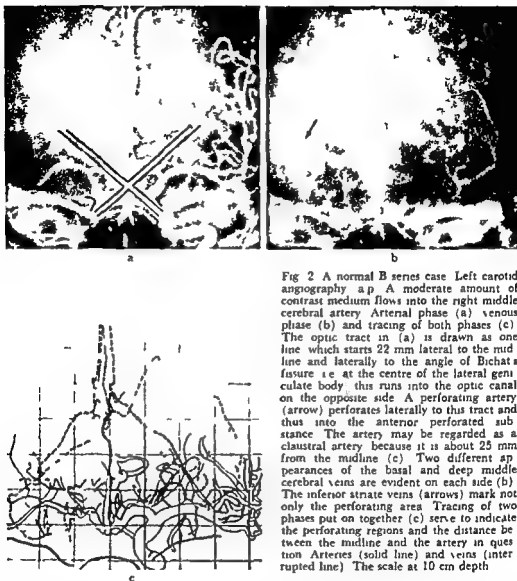


Fig 2 A normal B series case Left carotid angiography a p A moderate amount of contrast medium flows into the right middle cerebral artery Arterial phase (a) venous phase (b) and tracing of both phases (c) The optic tract in (a) is drawn as one line which starts 22 mm lateral to the midline and laterally to the angle of Bichat's fissure i e at the centre of the lateral geniculate body this runs into the optic canal on the opposite side A perforating artery (arrow) perforates laterally to this tract and thus into the anterior perforated substance The artery may be regarded as a claustral artery because it is about 25 mm from the midline (c) Two different appearances of the basal and deep middle cerebral veins are evident on each side (b) The inferior striate veins (arrows) mark not only the perforating area Tracing of two phases put on together (c) serve to indicate the perforating regions and the distance between the midline and the artery in question Arteries (solid line) and veins (interrupted line) The scale at 10 cm depth

artery and the midline measured directly Some errors may arise because the width of the third ventricle is not evaluated in the angiogram The distance from the insular surface of the lenticulostriate arteries may also be identified (Fig 2b) Small veins draining the surface of the insula and basal ganglia are landmarks favourable for indicating the exact border of these structures (WOLF & HUANG 1963) so that they sometimes cross perforating arteries (Fig 2 c) the latter must have perforating points

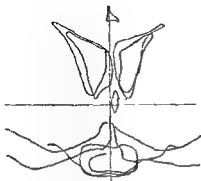


Fig 3



Fig 4

Fig 3 The contour of the base here lenticulostriate arteries perforate is traced according to the three sections of plates 5 and 6 LXXII in SCHALTENBRAND & BAILEY's atlas

Fig 4 The drawing is outlined from plates 47-49 LXXVIII of the atlas and the three reference points for a coordinate of the thalamus are indicated. Point A is at the most anterior bulging point of the anterior thalamic tubercle. Point B corresponds to the highest point of the interpeduncular fossa. Point C indicates both the pulvinar and superior tectal contours as they cross in the lateral projection. Points B and C may be movable. Figures on each side of the triangle are four times magnified. The extension of the ventral posterior nucleus (Vp) is projected and indicated by an interrupted line.

The optic tract as well as the optic nerve are located almost on a line drawn from the optic canal to the centre of the lateral geniculate body on the opposite side. In a frontal section of the atlas (LXXVIII plate 57) the centre of the lateral geniculate body lies about 22 mm lateral to the midline and occupies the most lateral angle of Bichat's fissure. Although the tract actually describes a slight reverse curve (LXXVIII plates 36-40) with its maximum at the chiasma the course may be plotted as a line. Arteries that perforate laterally to this tract are considered as lateral lenticulostriates because most perforate the anterior perforate area (SUZUKI 1961; WOLF & HUANG 1963) (Fig 2 a).

Frontal sections of SCHALTENBRAND & BAILEY's plates (LXXVII plates 5 and 11) when superimposed mark the perforating area and its slope in this projection in carotid angiography. At any rate a group of basal ganglia arteries may sometimes indicate the surface and slope of their perforating area by their course in the cisterns.

*Contribution of the method to the diagnosis of a pathologic condition of the thalamus.* The application of the stereotaxic method in the diagnosis of a pathologic condition of the thalamus demands some consideration being paid to other reference points and a coordinate suitable to the angiography. A few reference points may be determined by encephalography. Pathologic changes are however,

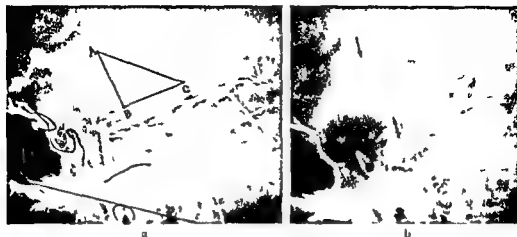


Fig. 5 A normal case of the A series. a) Arterial phase: Unit C is distinct in the film while Unit A is blurred. b) Prints A and B (arrows) are ascertained in the venous phase: the tracing of these points upon the film (c) forms the thalamic triangle ABC.

likely to invade and confuse the reference points themselves, so that they may be not so clear as such points in the angiograms. Vertebral angiography will demonstrate numerous arteries as well as veins that mark the configuration of the thalamus. The authors have consequently recorded certain fixed reference points in lateral vertebral angiograms and produced a new coordinate for the diagnosis.

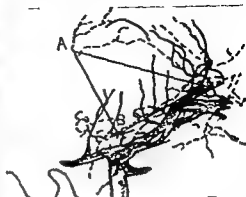
Both posterior medial and lateral choroid arteries approach each other (SUZUKI 1961; WOELSCHLAFFER & WOELSCHLAFFER 1966) as they reach the anterior end of the thalamus. The most anteriorly bulging part of the anterior thalamic tubercle (YAKOVLEV 1969) is here defined and is also identified by the venous channel around the foramen of Monro (JOHANSSON 1954; WOLF & HUANG 1961; GIUDICELLI & SALAMON 1970). This reference point in the angiogram is called point A (Fig. 5a, b). When examined in the plate of the atlas (I XXVIII, plate 42), each point A is about 4 mm apart from its fellow of the other side (Fig. 1). The lateral projection in the B series always presents the c as a single point A (Fig. 6a).

Both posterior choroid arteries run in the posterior and dorsal parts of the metencephalon and diencephalon (GALLOWAY & GREITZ 1960; SUZUKI 1961; OKAZAKI 1961). They are seen to cross in the lateral film at a point where in the lateral encephalogram the pulvinar crosses the upper quadrigeminal plate. This is called point C and when identified (I XXVIII, plates 42—49) presents a deviation amounting to a maximum of 3 mm in the direction of AC (Fig. 1).

The anterior pontine encephalic vein delineates the exact border of the inter



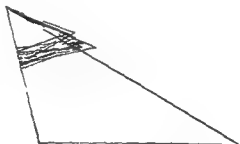
a



b

Fig 6 A normal case of the B series a) Lateral venous phase superimposed upon the scale at the midplane. Points A and B are clearly indicated (arrows) b) The triangle is formed on the tracing

Fig 7 Thalamic triangles of 17 normal cases of the A series superimposed upon the four times magnified standard triangle. Such is the same as that in fig 4. Magnification in these cases is not reduced.



peduncular fossa (HUANG et coll 1968 BRADAC 1970) the highest point of the roof forms point B. If the anterior pontomesencephalic vein does not completely lie at the midline, some information about point B may be given by determining the junction between the cisternal and parenchymal parts of the thalamoperforate artery (HARA & FUJINO 1966) point B must lie at the midline.

Connecting these three reference points of the thalamus triangle ABC is formed in the lateral vertebral angiogram and reflects the size and the shape of the thalamus. The triangle was plotted in 17 cases of the A series with normal vertebral angiography upon the standard but four times magnified triangle (Fig 7) as in Fig 4. Because of the varying magnification of the A series, these films were not reduced, and only the proportions of the triangles were calculated. The length of each side was examined in 5 cases of the B series (Fig 6). As observed at both examinations, most of these cases have a thalamus the length of the side AB of which deviates from the standard in the atlas. The authors have also



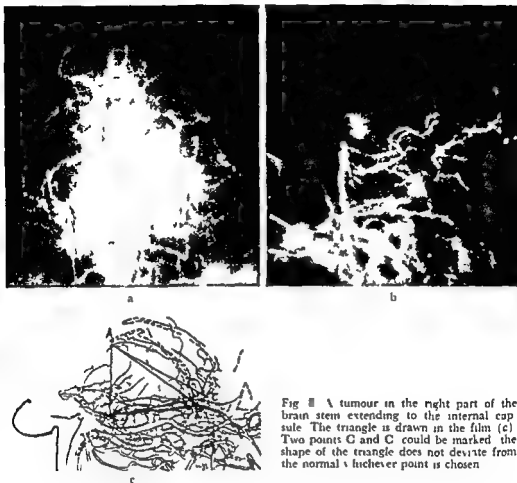


Fig 3 A tumour in the right part of the brain stem extending to the internal capsule. The triangle is drawn in the film (c). Two points G and C could be marked; the shape of the triangle does not deviate from the normal whichever point is chosen.

estimated the triangle of three normal cases in a Swedish hospital (see acknowledgement) and ascertained that the  $\epsilon$  triangles had similar measurements. The side AB of an average triangle therefore appears to be longer than the standard length 15 mm of the material LXXVIII; the average value of cases of the B series is about 20 mm. From the present material and other reports (VAN BUREN & MACCUBBIN 1962) it appears that the side AB of the plates (LXXVIII, plates 42–43) seems to be affected by more shrinkage than the other two sides.

It is fortunate for the application of the method to the diagnosis that the posterior ventral nucleus of the thalamus (HASSLER 1959; TREV *et coll.* 1969) occupies a larger portion of the line AC (Fig. 4). If any expansive process should invade this nucleus, the line AC becomes longer, resulting in a triangle



Fig 9 a) Tumour invading the right thalamus with many pathologic vessels b) The sides AB and AC of the triangle are considerably lengthened

deviating from the normal. The destruction of the nucleus is said to be clinically manifest by loss of tactile sense and other characteristic symptoms (WALKER 1959, YAKOVLEV 1969).

### Case reports

*Case 1 (of the 4 series)* Male aged 21 with motor disturbances of the left hand for about six months had developed diplopia, hoarseness and ptosis of the right eyelid. He dragged the left foot in walking. Clinical examination demonstrated right oculomotor palsy, hemiplegia with left facial nerve palsy, bilateral miotic pupils, disturbed accommodation of both eyes and pathologic reflexes of the left leg. Vertebral angiography (Fig 8 a, b) suggested a right thalamic tumour although the clinical signs indicated no or little impairment of the posterior ventral nucleus.

The triangle ABC of this case appeared to be of normal proportions (Fig 8 c). This excluded an invasion of the posterior ventral nucleus and confirmed the clinical examination. It was assumed that the neoplasm invaded the right internal capsule and more lateral structures.

*Case 2 (of the 4 series)* Male aged 26 who for 15 months had had motor disturbances of the left arm, hand and fingers with eventual partial paralysis of the left hand. His gait had become staggering for a fortnight. Neurologic examination demonstrated left hand tremor, left facial paresis and hypersensitivity of the skin of the left side of the body. The bilaterally positive Babinski reflexes disappeared soon after Torkildsen's shunt operation.

Vertebral angiography revealed pathologic vessels projected on the right thalamus (Fig 9 a). Contrary to Case 1 the thalamic triangle had changed in its proportions as well as in the length of each side (Fig 9 b). This indicated not only the extent of the tumour but also its precise location as invading the right posterior ventral nucleus.

### Discussion and Conclusion

The measurement of the neural structure does not include the evaluation of neural function. However the normal neural structures especially the basal ganglia, in their full development neither alter in size nor in shape. This is the reason why stereotaxic surgery is justified in locating the objective after the measurement of the target in roentgen films. This method of measurement may also be applied for the diagnosis if the investigation of the basal ganglia arteries in a living subject or the precise location of a basal ganglia tumour (TALAIRACH et coll. 1956) has been attempted. Angiographies are also able to indicate landmarks as well as a coordinate to the basal ganglia although the signs are somewhat different from those in encephalography.

The thalamus may be depicted as a triangle by connecting the three points mentioned in a lateral vertebral angiogram. If this triangle were estimated in many more cases a new diagnostic evaluation as to whether the thalamus of a case had a standard configuration or not would develop. Because the posterior ventral nucleus occupies a larger portion of one line of this triangle any change in the line is certainly an indication of a process invading the nucleus.

### Acknowledgement

The author takes this opportunity of thanking Prof. Torngy Greitz of the Department of Neuroradiology, Karolinska Spjukhuset, Stockholm, for permitting him to measure certain normal cases in his department.

### SUMMARY

The basal ganglia may be measured by determining reference points in angiograms. The application of this stereotaxic method in normal subjects promises some advantages by identification of the basal ganglia arteries. The measurement and configuration of the thalamus may also be applied in the precise diagnosis of a neoplasm of the thalamus.

### ZUSAMMENFASSUNG

Die Basalganglien können durch Bestimmung von Referenzpunkten in den Angiogrammen gemessen werden. Die Anwendung dieser stereotaktischen Methode bei normalen Individuen verspricht einige Vorteile durch die Identifizierung der Arterien der basalen Ganglien. Die Messung und Konfiguration des Thalamus kann ebenfalls verwendet werden um die genaue Diagnose eines Neoplasmas des Thalamus zu stellen.

## RÉSUMÉ

Il est possible de mesurer les noyaux gris centraux en déterminant des points de référence sur les angiographies. L'application de cette méthode stéréotaxique chez des sujets normaux semble permettre l'identification des artères des noyaux gris centraux. La mesure et la configuration du thalamus peuvent aussi être appliquées au diagnostic précis des tumeurs du thalamus.

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## TUMEURS DES NOYAUX GRIS CENTRAUX

Etude neuro radiologique avec verification anatomique

par

J LFORE J E PAILLAS M DUFOUR B ALLIEZ et A DEBAENE

Les 57 observations sur lesquelles repose notre travail ont toutes ete verifiees soit par intervention chirurgicale, soit par necropsie

Nous avons elimine les tumeurs lobaires avec extension secondaire aux noyaux gris ainsi que les tumeurs a point de depart ventriculaire pour ne considerer que les processus expansifs developpes aux depend des noyaux gris centraux thalamus noyau lenticulaire et noyau caude

Les caracteres angiographiques de ces tumeurs et les notions topographiques qui en decoulent reposent sur la dualite de leur vascularisation, qui depend a la fois du systeme vertebrel et du systeme carotidien comme l'avaient deja bien montre les travaux anatomiques anciens, repris par LAZORTHES

Quant aux signes ventriculaires ils ont longtemps constitue le seul element de diagnostic de ces tumeurs

*Etude histologique du materiel* Sur les 57 cas verifies anatomiquement 55 (26 tumeurs benignes et 29 tumeurs malignes) ont fait l'objet d'une etude histologique : 1 tuberculome 1 tumeur sarcomateuse, 1 kyste epidermoide 1 medullo-epitheliome 2 metastases 8 astroblastomes 10ependymogliomes (5 benignes 5 malins) 12 glioblastomes et 19 astrocytomes

Il existe un nombre a peu pres egal de tumeurs benignes et de tumeurs malignes

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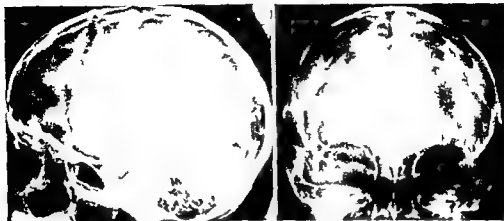


Fig. 1 Volumineuse calcification d'un astrocytome thalamique associe a des signes d'hypertension intra crânienne

*La vascularisation du thalamus* est assurée par des groupes artériels issus de la communicante postérieure de la cérébrale postérieure et des artères choroïdiennes postérieures. Ces groupes sont destinés aux parties antéro inférieure, postérieure et supérieure du thalamus.

*Le drainage veineux* est assuré par le système veineux profond et en particulier par la veine cérébrale interne à partir de la veine thalamique de la veine thalamo striée et de la veine caudée.

Ces notions angiographiques corroborent la classification topographique constatée par l'étude anatomique des tumeurs, qui nous permet de l'adopter pour leur étude angiographique.

### *Tumeurs postérieures*

*Examen standard* Les calcifications des tumeurs des noyaux gris centraux sont exceptionnelles : nous avons observé un cas de tumeur thalamique calcifiée chez une petite fille de 7 ans pour laquelle le diagnostic de tératome ou de pinealome avait été porté. L'étude histologique a montré qu'il s'agissait d'un astrocytome thalamique (Fig. 2). Signes d'hypertension intra crânienne : disjonction des sutures crâniennes et accentuation des impressions digitales.

### **Signes angiographiques**

#### *L'angiographie vertébrale*

L'angiographie vertébrale permet d'obtenir les renseignements les plus caractéristiques dans cette localisation tumorale (Fig. 3).



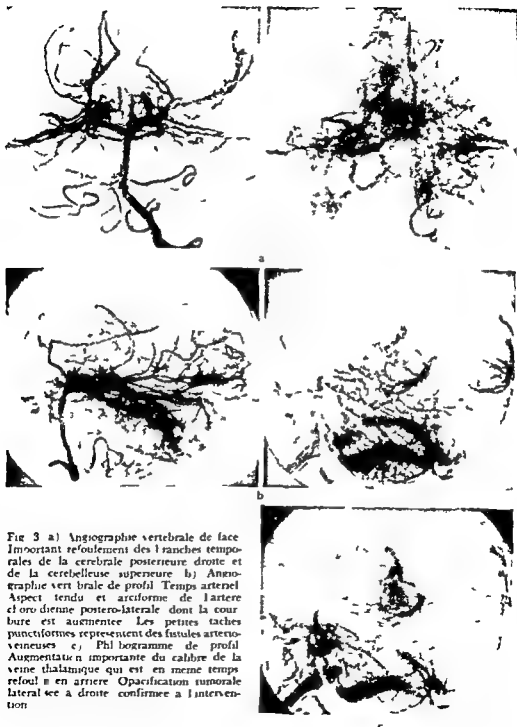


Fig 3 a) Angiographie vertébrale de face. Important refoulement des branches temporales de la cérébrale postérieure droite et de la cérébelleuse supérieure. b) Angiographie vertébrale de profil. Temps artériel. Aspect tendu et arciforme de l'artère carotidienne postéro-latérale dont la courbure est augmentée. Les petites taches punctiformes représentent des fistules artério-veineuses. c) Phlebogramme de profil. Augmentation importante du calibre de la veine thalamique qui est en même temps refoulée en arrière. Opacification tumorale latérale à droite confirmée à l'intervention.

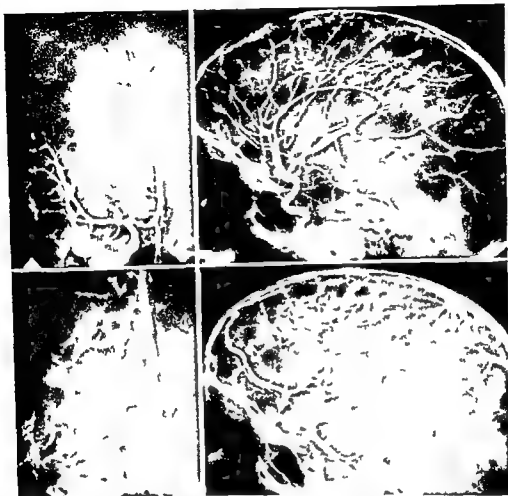


Fig 4 Angiographie carotidienne. Pas de modification des artères lenticulo-striées. Aspect cassé de l'artère sylvienne. Signes d'hydrocéphalie avec déplacement des pericallosales et de la veine de Galien.

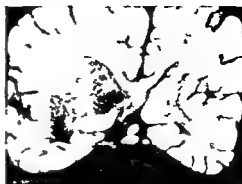
*Les artères (de profil)* Les artères choroïdiennes postéro-latérales sont étirées, déroulées et refoulées en arrière, leur courbure générale est agrandie. Par contre, la choroïdienne postéro-interne est déplacée plus tardivement et dans le même sens.

Les artères perforantes thalamiques peuvent être augmentées de volume, elles sont étirées et verticalisées, cette modification s'observe surtout sur les pédicules antérieurs issus de la communicante postérieure.

Dans certains cas, il existe des signes directs d'opacification tumorale avec fistules artério-veineuses à caractère punctiforme.



a



b

Fig 3 a) Déplacement arciforme de la veine thalamo-striée et refoulement de l'ampoule de Galien b) Coupe anatomique Tumeur du thalamus expliquant les signes veineux

L'existence de telles images permet de prévoir de la nature histologique maligne de la lésion.

*Le phlebogramme (de profil)* montre un refoulement postérieur des veines choroïdiennes et thalamiques dont le calibre peut être augmenté.

*De face* Les signes artériels sont beaucoup moins évidents surtout à un stade précoce parfois, cependant on peut observer une ou deux artères thalamiques bordantes par contre à un stade plus avancé, lorsque la tumeur gagne les pedoncules on observe un abaissement de la fourche peripédunculaire des cérébrales postérieures et des cérébelleuses supérieures. Cet aspect est surtout mis en évidence en incidence directe antéro-postérieure.

#### *L'angiographie carotidienne*

*Les artères de face*, Il n'y a habituellement, pas de retentissement artériel dans les tumeurs postérieures. Lorsque les artères lenticulo-striées sont déplacées, cela traduit une extension antérieure de la tumeur. L'artère pericalléuse n'est pas déviée. Par contre les artères insulaires sont refoulées en dehors et verticalisées.

Fig 4

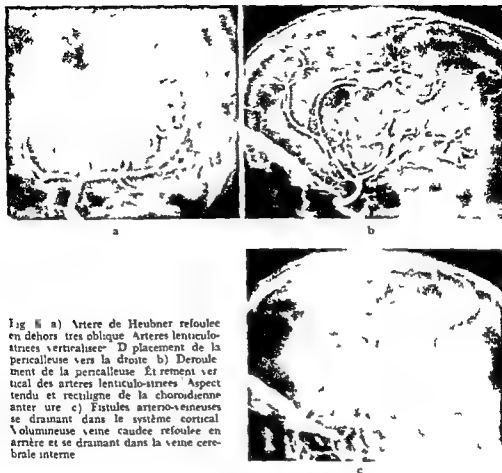


Fig 5 a) Artere de Heubner refoulee en dehors tres oblique Arteres lenticulo-striees verticalisees Dplacement de la pericalleuse vers la droite b) Deroulement de la pericalleuse Etirement vertical des arteres lenticulo-striees Aspect tendu et rectiligne de la choroidienne anterieure c) Fistules arterio-veineuses se drainant dans le systeme cortical Volumineuse veine caudee refoulee en arriere et se drainant dans la veine cerebrale interne

*Le phlebogramme (de face) profond* montre des modifications importantes interessent l'ampoule de Galien et la veine cerebrale interne qui sont largement refoulees du cote oppose la veine thalamo-striece presente un refoulement et un soulèvement arciforme reproduisant exactement le developpement tumoral (Fig 5)

*Les arteres (de profil)* Le déplacement de l'artere sylvienne est assez caracteristique traduisant le déplacement transversal du vaisseau de dedans en dehors, dans la vallee sylvienne Il peut presenter alors un aspect « casse » en « ligne brisee »

L'artere pericalleuse lorsqu'il s'agit d'une tumeur volumineuse peut etre le siege d'un deroulement de sa partie posterieure

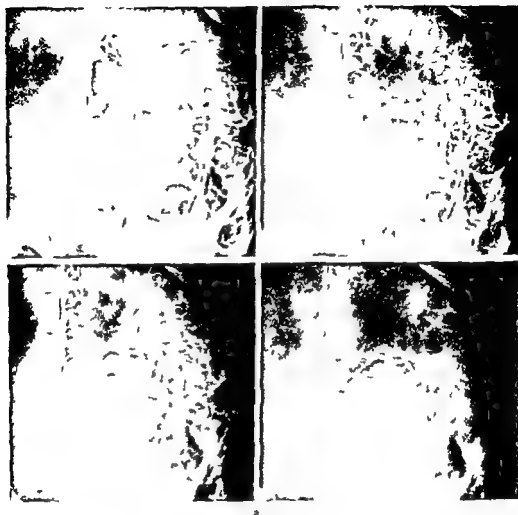
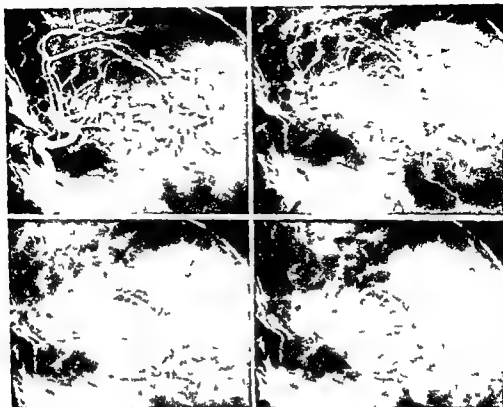


FIG. 7 (Pour légende voir la page opposée)

L'artère choroidienne antérieure est tendue et déroulée dans sa portion plexuelle.

Le phlebogramme (de profil) profond montre un soulèvement de la veine cérébrale interne et l'ouverture de l'angle veineux, par écartement prédominant de la veine thalamo-striée. La veine basale est abaissée dans sa portion proximale. Ces deux déplacements veineux opposés se traduisent par un écartement des vaisseaux.



b

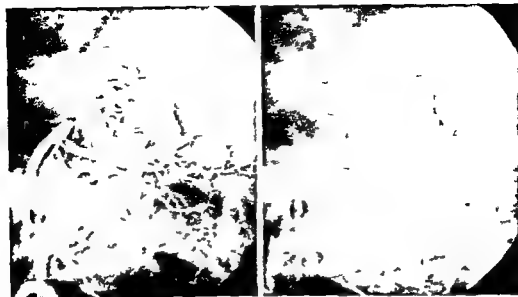
Fig 7 Angiographie carotidienne a) De face b) De profil Volumineuse tumeur vascularisée par des pédicules provenant de l'artère sylvienne et de l'artère choroidienne antérieure neovaisseaux pathologiques anarchiques. Très faible déplacement de l'axe cérébral antérieur témoignant de la prédominance postérieure de la tumeur

### *Tumeurs antérieures*

Ces tumeurs sont essentiellement mises en évidence par l'angiographie sélective de la carotide interne (Fig 6)

*Les artères (de face)* Le retentissement porte surtout sur les artères lenticulo-striées qui peuvent être étirées tendues verticalement ou, au contraire, refoulées en dehors elles tendent à s'écarter de la ligne médiane et à se rapprocher des branches insulaires de la sylvienne

A un stade plus avancé l'augmentation de volume de la tumeur entraîne un refoulement de la pericalléuse du côté opposé et de la sylvienne en dehors



2

Fig. 8. — Pour l'étude voir la page opposée

Lorsque la tumeur s'étend vers le bas la choroïdienne antérieure peut être refoulée en dehors.

*Le phlebogramme (de face).* Au stade de début le phlebogramme profond n'est pas modifié.

Lorsque la tumeur s'étend vers l'arrière et qu'elle atteint le corps du noyau caudé, la veine thalamo-sinéc est refoulée en haut et en dedans décrivant une image arciforme, quant à la veine cérébrale interne elle est très déplacée du côté opposé.

Lorsque les veines septales et caudées sont injectées elles peuvent être déviées en dedans si la tumeur intéresse la tête du noyau caudé.

*Les artères (de profil).* Les artères lenticulo-sinéc difficiles à discerner sont tordues verticalement et tendues.

Lorsque la tumeur s'étend au thalamus, les artères thalamo-perforées antérieures sont déviées vers l'arrière.

En cas d'extension tumorale antérieure on observe un renflement sur l'artère perforante qui est déroulée.

En cas d'extension basale l'artère choroïdienne antérieure est tendue et rectifiée.

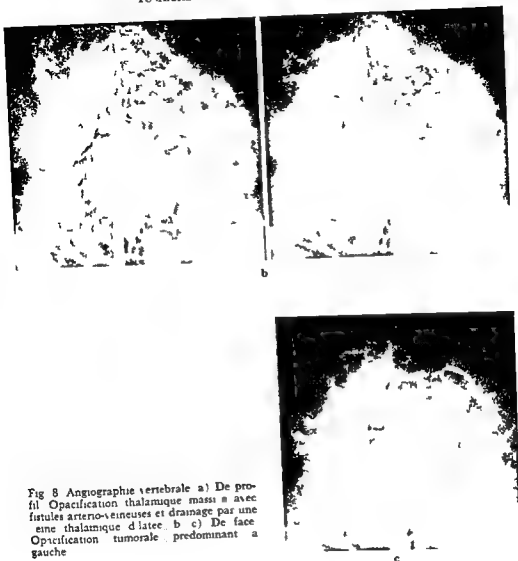


Fig 8 Angiographie vertebrale a) De profil Opacification thalamique massive avec fistules artéro-veineuses et drainage par une veine thalamique dilatée. b c) De face Opacification tumorale prédominante à gauche

*Le phlebogramme (de profil) montre un soulèvement de la veine cérébrale interne et de l'angle veineux*

Il peut exister des fistules artéro-veineuses avec drainage à partir de la veine caudale dans le système veineux profond

#### *Tumeurs globales*

Ces tumeurs s'opacifient à la fois par le système carotidien et par le système vertébral (Figs 7 8)



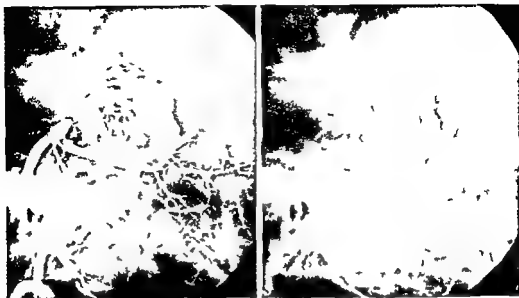


Fig. III (Pour légende voir la page opposée)

Lorsque la tumeur s'étend vers le bas, la choroïdienne antérieure peut être refoulée en dehors

*Le phlebogramme (de face)* Au stade de début, le phlebogramme profond n'est pas modifié

Lorsque la tumeur s'étend vers l'arrière et qu'elle atteint le corps du noyau caudal la veine thalamostriée est refoulée en haut et en dedans, décrivant une image arciforme quant à la veine cérébrale interne, elle est très déplacée du côté opposé

Lorsque les veines septales et caudales sont injectées elles peuvent être déviées en dedans si la tumeur intéresse la tête du noyau caudal

*Les artères (de profil)* Les artères lenticulo-striées difficiles à discerner sont tirées verticalement et tendues

Lorsque la tumeur s'étend au thalamus les artères thalamo perforées antérieures sont déplacées vers l'arrière

En cas d'extension tumorale antérieure on observe un renflement sur l'artère péricalluse qui est déroulée

En cas d'extension basale, l'artère choroïdienne antérieure est, soit tendue et rectiligne soit abaissée

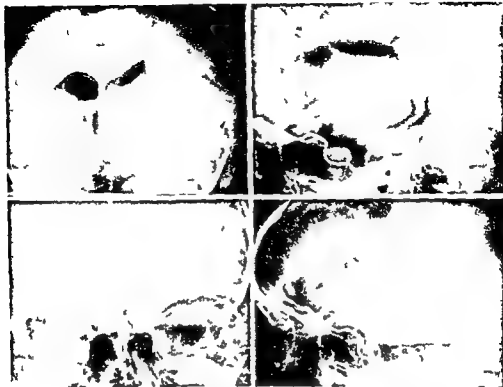


Fig 10 Tous les signes ventriculaires et cisternaux se retrouvent : écartement de carrefour, abaissement de la corne temporale, retoulement du troisième ventricule avec aspect en « croissant de lune » et décalage des cisternes retro-pulvinariennes.

troisième ventricule et par une déformation caractéristique en « croissant de lune ».

Le carrefour ventriculaire est ouvert et décalé en hauteur par rapport au côté sain. La corne temporale est abaissée et comprimée.

Il existe aussi, dans certains cas, un retentissement sur les cisternes retro-pulvinariennes qui sont décalées l'une par rapport à l'autre (Fig 10).

L'iodo ventriculographie est indiquée lorsqu'il existe un blocage au niveau du troisième ventricule ou de l'aqueduc qui « oppose » la progression du contraste gazeux.

Dans ces cas, il est souvent difficile de faire le diagnostic entre tumeur thalamique et tumeur du troisième ventricule (Fig 11).

Il semble que cet examen soit plus souvent indiqué chez l'enfant que chez l'adulte.



Fig 9 Tumeur de la tête du noyau caudé droit envahissant le plancher de la corne frontale qui est laminée. Déplacement du septum du côté opposé. De profil on distingue l'image de la tumeur dans le plancher de la corne.

Suivant la prédominance de leur localisation antérieure ou postérieure on constate une prédominance du système carotidien ou du système vertébral.

Étant donné l'importance habituelle du volume de ces tumeurs et leur double irrigation il est habituel d'observer une vascularisation pathologique très accusée.

### Signes ventriculaires

*Les signes ventriculographiques* sont très fidèles et ont représenté le seul élément du diagnostic neuro-radiologique des tumeurs des noyaux gris centraux, pendant de nombreuses années.

Ces signes sont, dans l'ensemble, bien connus, nous ne ferons que les rappeler brièvement.

L'hydrocéphalie ventriculaire peut s'observer lorsque la tumeur comprime l'un ou les deux trous de Monro ou le troisième ventricule.

Dans d'autres cas les déformations des parois des ventricules indiquent la localisation tumorale.

Les tumeurs antérieures de la tête du noyau caudé modifient essentiellement le plancher de la corne frontale qui est surélevé ou envahi ainsi que la partie antérieure du troisième ventricule. Le septum peut être déplacé (Fig 9).

En ce qui concerne les tumeurs postérieures essentiellement thalamiques les signes ventriculaires sont principalement constitués par un déplacement du

## ZUSAMMENFASSUNG

Eine Untersuchung von 51 Fällen mit Tumoren der zentralen Kerne des Gehirns wurde durch Operation oder Nekroskopie bestätigt. Diese Arbeit ermöglichte die topographische Unterscheidung zwischen drei Gruppen von Tumoren: einer anterior gelegenen, die den Nucleus caudatus und den Nucleus lentiformis umfasst; einer posterior gelegenen, die den Thalamus umfasst; und einer alle Gebiete umfassenden Gruppe.

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## VENENBILDER BEI AKUSTIKUSNEURINOMEN

von

R. LEHMANN

Über den großen diagnostischen Wert und die weitestgehende Ungefährlichkeit der Vertebralisangiographie bestehen heute keine Meinungsverschiedenheiten mehr. Gegenwärtig wird vielerorts an der verfeinerten Diagnostik der Vertebralisangiographie gearbeitet.

In einer Nachuntersuchung von 80 operativ bestätigten Tumoren der hinteren Schädelgrube stellten wir 1966 fest, daß die Vertebralisangiographie um so sichere Resultate liefert, je weiter lateral die raumfordernden Prozesse lokalisiert sind.

Der Wert der Vertebralisangiographie für Akustikusneurinome ist seit langem bekannt. Zur Diagnostik werden aber im wesentlichen die arterielle und — bei pathologischen Gefäßen — die kapillare Phase herangezogen. Die venöse Phase wurde lange Zeit wenig berücksichtigt. Nach intensivem Studium der angiographischen Venenbilder der hinteren Schädelgrube durch u. a. JOHANSSON, WACKENHEIM, BRADAC et coll., ROSA & VIALE, HUANG & WOLF sind jetzt die pathologischen Abweichungen im Phlebogramm der hinteren Schädelgrube exakter zu definieren.

Wir haben 33 Vertebralisangiogramme mit den operativ oder autopsisch gesicherten Befunden von Akustikusneurinomen nachträglich analysiert. Von den

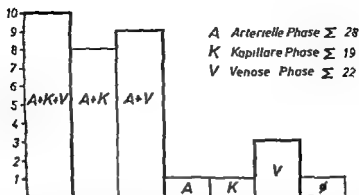


Abb 1 Diagnostische Relevanz der Phasen im Vertebralisangiogramm von 33 Akustikusneurinomen. Symptome für einen raumfordernden Prozess waren 32 mal zu finden, davon 22 mal in der venösen Phase, die 3 mal allein diagnostisch relevant war. Ein Fall bot in keiner Phase angiographische Symptome.

33 Angiogrammen hatten 28 in der arteriellen, 19 in der kapillaren und 22 in der venösen Phase Symptome, die für Kleinhirnbrückenwinkeltumoren sprachen. Die diagnostische Bedeutung der einzelnen Phasen wird in Abb 1 gegeben, wo auch demonstriert wird, daß in den meisten Fällen 2 oder 3 Phasen zur Diagnostik von Kleinhirnbrückenwinkeln herangezogen werden können. Drei mal war die venöse Phase allein ausschlaggebend, und je einmal war die arterielle oder die kapillare Phase allein diagnostisch bedeutungsvoll. In einem einzigen Fall fanden wir in keiner Phase pathologische Zeichen.

Das kleinste von uns angiographisch diagnostizierte Akustikusneurinom war kirschgroß.

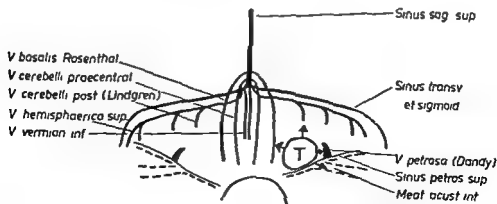


Abb 2 Venenschema der Fossa posterior in sagittaler Projektion mit Angabe der Druckrichtung bei Akustikusneurinom (Modifiziert nach WACKENHEIM).



Abb. 3 Anhebung und Lateralisierung der linken V. petrosa durch ein Akustikusneurinom

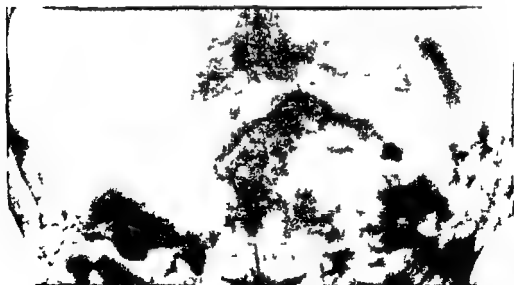


Abb. 4 Angiomartig gefäßer Brenner-Tumor mit Randvenen und Lateralisierung der linken V. petrosa sowie Kompression des linken Sinus petrosus superior

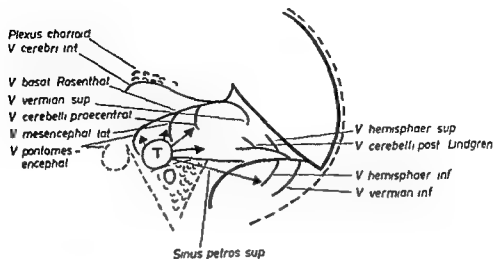


Abb 5 Venenschema der Fossa posterior im seitlichen Bild bei Akustikus neurinom. (Modifiziert nach WACKENHEIM)

### Befunde im Phlebogramm

Am bedeutungsvollsten ist die Beurteilung der V petrosa. Sie liegt in der Nähe des Porus acusticus internus und wird durch raumfordernde Prozesse in diesem Bereich lateralisiert und angehoben. Bei großen raumfordernden Prozessen wird sie allerdings komprimiert und ist nicht zu beurteilen. Der Sinus petrosus superior ist bei optimaler Projektion und im Subtraktionsbild gut erkennbar und verschieden stark ausgebildet. Er kann umchrieben verlagert werden. Von großen raumfordernden Prozessen kann er medial oder total komprimiert werden. In der superioren Venengruppe sind eine Anhebung bzw. Medialisierung der V basalis Rosenthal, eine Anhebung der V ponto-mesencephalica und eine Medialisierung und Anhebung der V cerebelli praecentralis bedeutungsvoll. Bei großen raumfordernden Prozessen können diese Venen infolge Kompression ebenfalls angiographisch nicht darstellbar sein.

Die V hemisphaerica superior kann bei größeren Tumoren angehoben sein. Eine Verlagerung der inferioren Venengruppe findet sich nur bei großen raumfordernden Prozessen. Für die Beurteilung des seitlichen Bildes ist allerdings zu beachten, daß die Venen nicht in der Mittellinie verlaufen. Sie können deshalb projektorisch mehr oder minder weit von der Kalotte entfernt sein. Eine Anlagerung der Venen an die Kalotte kann deshalb nur in Verbindung mit einer Ausspannung und weiteren Zeichen für einen großen raumfordernden Prozeß gewertet werden. Die Druckrichtung auf diese Venen ist aber aus der Venen





Abb 6 Anhebung der  $\vee$  basalis der  $\vee$  pontomesencephalica und der  $\vee$  mesencephalica lateralis sowie Occipitalverlagerung der  $\vee$  cerebelli präcentralis. Verlagerung durch ein sehr großes Akustikusneurinom der inferioren Venengruppe und des Torkildsen-Schlauches durch den Operationsdefekt

verlagerung durch einen Operationsdefekt der Okzipitalschuppe hindurch zu erkennen

Im Gegensatz zur arteriellen Phase können bei größeren raumfordernden Prozessen die Venen der Tumorregion durch Kompression nicht gefüllt werden. Dieses Symptom ist dann nur als indirektes Zeichen in Verbindung mit anderen positiven Zeichen zu werten. Voraussetzung für die Bewertung derartiger Nichtfüllungen ist aber eine optimale Röntgentechnik, eine genügende Kontrastmitteldosis und ein regelrechter Kontrastmittelabstrom.

Nach Operation ist die verminderte oder fehlende Venenzeichnung im Operationsgebiet diagnostisch nicht heranzuziehen.

Differentialdiagnostisch kommen neben Neurinomen der anderen Hirnnerven dieser Region Meningeome Sarkome und seltene Tumoren in Betracht. Weiterhin kommt bei Venenverlagerungen eine Arachnoiditis mit oder ohne Zysten in Frage.

Von röntgentechnischer Seite muß betont werden, daß gefäßarme Tumoren und kleinere Gefäße bedeutend besser zu beurteilen sind, wenn die Serien in Uniplanstechnik aufgenommen sind. Weitere Parameter sind eine optimale Einstellungstechnik, Belichtungstechnik und Filmverarbeitung, Fokussierung der Röhre, Röhrenspannungen bis 80 kV sowie zeichenschärfe Folien und ein genügend langes Serienprogramm.

Methodisch ist die selektive Füllung der A. vertebralis zu bevorzugen. Eine

Kontrastmittelmenge von 8 bis 10 ml pro Injektion ist zur guten Darstellung der Venen notwendig. Falls nicht besondere Gründe entgegenstehen, sollte die Seite des Tumors katheterisiert werden. Dadurch ist eine bessere Füllung der tumorseitigen A. cerebelli inferior posterior gegeben. Grundsätzlich ist die a.p. Serie diagnostisch bedeutungsvoller als die seitliche.

## ZUSAMMENFASSUNG

Dreiunddreißig Vertebraisangiogramme von Patienten mit bestätigten Akustikusneurinomen wurden nachträglich hinsichtlich der venösen Phase analysiert. Zweiundzwanzig Phlebogramme wiesen pathologische Veränderungen auf, die für Kleinhirnbrückenwinkel-tumoren sprachen. Die Venenverlagerungen werden einzeln besprochen. Kurze Hinweise auf methodisch-technische Fragen schließen sich an.

## SUMMARY

A retrospective analysis of 33 vertebral angiographies in patients with known neuromas of the auditory nerve is presented. The venous phase was the main subject of the investigation and 22 phlebograms disclosed changes typical of cerebello-pontine tumours: the venous displacements are reported in detail. Guide lines for the best technical approach are presented.

## RÉSUMÉ

L'auteur a analysé retrospectivement trente-trois angiographies vertébrales de malades atteints de neurinome acoustique confirmé en étudiant la phase veineuse. Vingt-deux phlébogrammes présentaient des altérations pathologiques qui étaient en faveur du diagnostic de tumeur de l'angle ponto-cérébelleux. L'auteur étudie séparément chacun des déplacements veineux. Il examine brièvement quelques questions de technique.

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## MULTIPLE PROGRESSIVE INTRACRANIAL ARTERIAL OCCLUSIONS

by

E V LESLIE and G J ALKER JR

A variety of occlusive cerebrovascular processes are encountered in young individuals where the etiology is often unclear. Efforts are made to exclude the coagulopathies, arteritis, sources of emboli, and more recently, the use of oral contraceptives (MASI & DUGDALE 1970). In the Caldwell lecture of 1968, TAVERAS described a group of young patients with occlusive cerebrovascular disease which appeared to represent a clinical entity, and he coined the term multiple progressive intracranial arterial occlusions. It seemed similar, if not the same, as that described heretofore only in the Japanese (KAWAKITA et coll 1965, KUDO 1968, LEEDS & ABBOTT 1966, NISHIMOTO & TAKEUCHI 1968). This prompted us to review a number of our angiograms showing occlusive disease of the young. Included among the various patterns were three patients whom we felt most probably represented instances of this same entity.

The clinical presentation may be characterized by severe headache, by subarachnoid hemorrhage, perhaps from the dilated transdural collaterals, alternating hemiplegias and occasionally changes in mood. Both sexes are affected and the age incidence in TAVERAS' series was from seven months to forty-two years; thus he referred to it as a syndrome of children and young adults.

Development of extensive transdural anastomoses may be associated with dilated meningeal grooves on conventional films. This may be especially discernible if the skull has been examined before.



Fig 1



Fig 2

Fig 1 Ap view of right carotid angiography with cross compression of the left carotid artery. Tapering of the internal carotid artery up to the stenosis of the supraclinoid portion. The pericallosal artery is almost completely occluded at its junction with the anterior communicating artery. The posterior cerebral artery filled directly from the internal carotid and slightly narrowed proximally. The major intracranial branches appear normal.

Fig 2 Ap view of left carotid angiography with essentially the same features as on the right side, but both pericallosal arteries filled from this side.

The silent angiographic features (TAVRAS) include the progressive bilateral narrowing of the supraclinoid portion of the internal carotid arteries and the anterior and middle cerebral arteries. Occasionally the posterior communicating and the posterior cerebral arteries are involved. The basilar artery may also show diffuse narrowing. Ultimately, occlusion of the distal internal carotid artery may occur on one or both sides. There is a tapering of the internal carotid artery from just distal to its origin to the level of the stenosis, a change which pre-



Fig 3 Lateral view of left carotid angiography demonstrates an infraclinoid aneurysm and normal appearance of the intracranial branches

ably is a manifestation of diminished flow in that artery. Particularly in complete occlusion of the proximal anterior and middle cerebral arteries a collateral flow through a plethora of perforating branches may occur (NISHIMOTO & TAKEUCHI, TAVERAS) and in the cases with basilar artery involvement there may also be discernible multiple transdural anastomoses: rete mirabile (MOUNT & TAVERAS 1977, TAVERAS).

One of our cases was of particular interest because of efforts made to exclude any vasoplastic element. This case will be presented in detail.

### Case report

A twenty four year old white female was admitted complaining of having had four or five episodes of bitemporal headache associated with numbness in the fingers of the left hand and the left trunk down to the waist. These had occurred during the two months before admission. With the attacks she would feel faint, warm, nauseated and somewhat dyspnoic. There was no loss of consciousness. The headache was constant and aching, not throbbing, and would come on suddenly, often after the neurologic attack.

On examination the salient features were slightly elevated blood pressure of 140/80 and a cardiac murmur which was felt to be functional by the cardiologists. Neurologically the patient was intact. FCG, chest films and conventional skull films were normal. EEG showed moderately severe slow wave abnormality in the right frontal area extending to the right central and temporal areas. Examination of the cerebrospinal fluid was entirely normal. Lupus erythematosus tests were negative as were anti nuclear antibodies. Sedimentation rate, blood sugar and several other laboratory examinations were normal. A biopsy of the right superficial temporal artery was histologically normal.

Encephalography revealed minimal diffuse cortical atrophy. Bilateral carotid angiography demonstrated symmetrical stenoses of the distal internal carotid arteries and abnormal

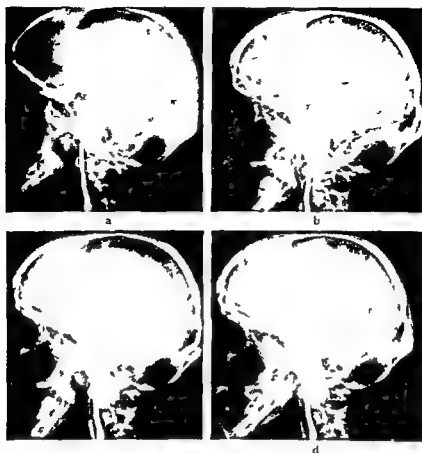


Fig. 4 Right lateral angiography demonstrating the stenosis: a)  $pCO_2$  23 mm and pH 7.45; b) 3%  $CO_2$  ( $pCO_2$  30 mm and pH 7.39); c) 10%  $CO_2$  ( $pCO_2$  41 mm and pH 7.33); d) 1 hour later after intrarterial injection of 25 mg of papaverine.

stretched appearance of all of the intracranial arteries (Figs 1-2). The carotid siphons generally appeared straightened and flattened on either side but there was no beading. An intraclinoid aneurysm was present on the left side (Fig. 3). The posterior cerebral arteries filled directly from the carotid artery on either side and were minimally narrowed. Both pericallosal arteries filled from the left side (Fig. 2).

Two months later the patient was readmitted because of some involuntary spasm of the left arm and a tendency of the left leg to buckle. The right carotid angiography was repeated with a view to excluding any possibility of vasospasm. On a lateral view exposed when the patient's  $pCO_2$  was 23 mm and the pH was 7.45 the stenoses are clearly evident (Fig. 4) and there is overall diffuse straightening of the vessels. The next injection was made with the patient breathing 3%  $CO_2$ . At this time the  $pCO_2$  had risen to 38 mm and the pH had dropped to 7.39. No change was evident in the vessels. The next injection was then made ten minutes after the patient commenced breathing 10%  $CO_2$ .

carbon dioxide. At this point the pCO<sub>2</sub> had risen to 41 mm and the pH had dropped to 7.33. The patient felt quite dyspneic at this point. Despite the profound vasodilatory effort the vessels remained unchanged. The last injection was made after the intra-arterial injection of 25 mg of papaverine; no change was seen.

Several months later the patient expired of a massive cerebrovascular accident at another institution where she was operated upon for hydronephrosis secondary to a uretero-pelvic obstruction. Nephroangiography and abdominal aortography had been essentially normal. Gross and microscopic examination revealed what appeared to be a small basilar artery with a recent thrombus therein. The right posterior cerebral artery contained a recanalized thrombus while the left posterior cerebral artery was patent. One small arteriosclerotic plaque was present in the left posterior cerebral artery more peripherally. The pathologist reported some intimal thickening in the internal carotid arteries with an abundance of dilated small arteries in the area of the posterior perforating arteries. No other specific change or inflammatory response was evident in any of the arteries. The prosector felt that the coronary arteries appeared somewhat small in size but histologically these as well as the remaining arteries throughout the body were normal.

### Discussion

This patient clearly had multiple progressive intracranial arterial occlusions without clear etiology. She did not develop the spectacular collaterals either at the base of the brain or through transdural anastomoses which others have reported.

Supraclinoid stenosis or occlusion is actually rare (SILVERSTEIN & HOLLIV 1963) and can have varying etiologies to include arteriosclerosis, systemic lupus erythematosus, spasm and subsequent thrombosis associated with intracranial aneurysms, embolus, dissecting aneurysm, tuberculosis and lues. In the case of multiple progressive intracranial arterial occlusions the process is essentially bilateral involving not only the supraclinoid portion of both internal carotid arteries but often the proximal portions of anterior, middle and posterior cerebral arteries. While this occlusive process of distal internal carotid arteries and the circle of Willis was originally described as restricted to Japanese, it seems apparent that the same or similar entity occurs in other races. The amount of pathologic material available is limited and despite having gross and pathologic material available in one patient, there is still no clear definition of the pathology or of its etiology. Vasospasm does not appear to play a significant role in this disease entity. It may well be that these patients do have congenitally small arteries but this is rather speculative. Our patients have not shown the extensive collateral formation via the perforating arteries or the transdural anastomoses, but we would feel that this perhaps represents a part of the spectrum of the disease. The distal internal carotid artery and the more proximal portions of the anterior, middle and posterior cerebral arteries had a rather stretched and abnormal appearance.



## SUMMARY

A review of our angiographic material revealed several cases which appear to conform with TAVERAS' criteria for multiple progressive intracranial arterial occlusions. One such case is presented in detail to show that there is no apparent vasospastic element involved in the supraclinoid stenoses and occlusions. This patient did not demonstrate the development of extensive collateral flow via either the perforating artery or the transdural anastomoses.

## ZUSAMMENFASSUNG

Eine Übersicht unseres angiographischen Materials liess verschiedene Fälle zutage treten die mit TAVERAS' Kriterien für multiple progressive intracraniale arterielle Okklusionen übereinzustimmen scheinen. Ein derartiger Fall wird im einzelnen vorgestellt um zu zeigen dass kein augenscheinliches vasospastisches Element an den supraclinoiden Stenosen und Okklusionen beteiligt sind. Dieser Patient zeigte nicht die Entwicklung einer extensiven Kollateraldurchblutung weder über die perforierende Arterie noch über den transduralen Anastomosen.

## RÉSUMÉ

L'examen de nos cas d'angiographie a montré plusieurs cas qui semblent répondre aux critères donnés par TAVERAS pour définir les occlusions artérielles intracrâniennes multiples progressives. Nous présentons un de ces cas en détail pour démontrer qu'il n'y a pas d'élément apparent de spasme vasculaire intervenant dans les sténoses susclinoidiennes ni les occlusions. Nous n'avons pas mis en évidence chez ce malade le développement d'une importante circulation collatérale soit par l'artère perforante soit par les anastomoses transdurales.

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## BORDERLANDS OF THE NORMAL AND ABNORMAL POSTERIOR INFERIOR CEREBELLAR ARTERY

by

M. THEODORE MARGOLIS and THOMAS H. NEWTON

The posterior inferior cerebellar artery (PICA) supplies the medulla the inferior portion of the fourth ventricle the inferior vermis the tonsils and the inferior aspects of the cerebellar hemispheres. The PICA and its branches are of great importance therefore in the diagnosis of lesions in the posterior fossa. Selective catheter techniques combined with magnification and subtraction permit a more detailed analysis of the PICA and its branches. The evaluation of these examinations depends upon a knowledge of the normal gross and roentgenographic anatomy of this vessel.

The origin course and distribution of the PICA vary greatly so that at times differentiation between the abnormal and the variant of normal is difficult for the neuroradiologist.

In this report we correlate the gross and angiographic anatomy of the PICA and emphasize the wide variations of normal. These variations are compared with the borderline abnormal pathologic changes. Particular attention is given to those segments of the artery that cause the most confusion. These are (1) the caudal loop — its relation to the diagnosis of tonsillar herniation (2) the occasional lateral course of the PICA around the tonsil (3) the choroid arch — its relation to the fourth ventricle and measurements of its normal position in lateral



Fig 1 Sagittal section of cerebellum with medulla removed to demonstrate the caudal loop or lateral medullary segment (—) posterior medullary segment (—) and supratonsillar segment or choroid arch (—) Choroid plexus in fourth ventricle (1) Lower pole of tonsil (2)



2a



2b

Fig 2 Normal variation of the caudal loop which extends down to the level of C1



Fig 3 a) Ap view of cerebellum pons and medulla showing the relation between tonsillar ( $\rightarrow$ ) and hemispheric ( $\leftrightarrow$ ) branches Right posterior inferior cerebellar artery (1) tonsils (2) b) Undersurface of cerebellum and tonsils Right posterior inferior cerebellar artery ( $\rightarrow$ ) tonsils (1) vermis (2) hemispheric branches ( $\leftrightarrow$ ) tonsillar branch ( $\rightarrow$ )

and half axial projections and in borderline displacement and ( $\pm$ ) the vermis branches — their measurements in the half axial projection, and differentiation from medial hemispheric branches

**Caudal loop** The caudal loop or lateral medullary segment (HUANG & WOLF 1969) often curves around the anterior margin of the lower pole of the tonsil (Fig 1). In the lateral projection the apex of the caudal loop in most instances lies above the foramen magnum. In 35 % of our normal cases, however, the caudal loop was seen to extend below the level of the foramen magnum. The average distance below the foramen magnum measured 5 mm, the maximum 20 mm (Fig 2) (MARGOLIS & NEWTON 1972). In these cases, the lower portion of the loop does not correspond to the inferior margin of the tonsil.

The angiographic diagnosis of tonsillar herniation has been considered difficult because of the many variations in the origin and course of the PICA (DILENCE & DAVID 1967, WOLF et coll 1962, GREITZ & SJOGREN 1963, TAVERAS & WOOD 1964, MARGOLIS and NEWTON 1971). The tonsils may herniate without displacing the caudal loop and conversely a low caudal loop may be present normally. The diagnosis of tonsillar herniation in the past has depended on the demonstration of downward displacement of the tonsillar branches of the PICA (GREITZ & SJOGREN 1963). These branches are often too small, however, to be recognized. A more reliable sign of herniation is the change in the hemispheric branches (MARGOLIS & NEWTON 1971). The separation of the PICA into its two main terminals, the tonsillohemispheric and vermis branches, occurs a short distance distal to the apex of the cranial loop (WOLF et coll 1962). The



Fig 4 Same specimen as in fig 3 a) Ap view of injected specimen. Normal undulating appearance of the hemispheric branches along the undersurface of the cerebellum ( $\rightarrow$ ) b) Ap projection of vertebral angiogram. The hemispheric branches are separated from other arteries and are easily identified in this view ( $\rightarrow$ )

tonsillohemispheric branch descends along the posterior margin of the medial aspect of the tonsil. It divides into tonsillar branches that have a variable course and hemispheric branches that extend downward and posterolaterally. The hemispheric branches supply the undersurface of the hemispheres as far as its lateral border and anastomose with the anterior inferior cerebellar artery and superior cerebellar artery. The site of branching of the tonsillohemispheric stem into tonsillar and hemispheric branches varies considerably (Fig 3). Because of their common origin, however, any downward displacement of the tonsil simultaneously stretches and pulls the hemispheric branches inferiorly. The hemispheric branches are best demonstrated in the ap projection (Fig 4). As the tonsils herniate they pull and stretch the adjacent hemispheric branches, carrying them down over the edge of the foramen magnum. In the lateral projection straightening may be seen of the hemispheric branches, but the stretching down over the edge of the foramen magnum is often obscured by the low cranial loop itself (Fig 5 a). In the ap projection (central ray  $5^\circ$  cephalad to orbitomeatal line) viewed tangentially at the foramen magnum, these stretched hemispheric branches are easily seen (Fig 5 b).

*Lateral course of the PICA around the tonsil.* On reaching the posterior margin of the medulla oblongata, the PICA ascends medial to the tonsil. It continues its ascent to the anterior aspect of the superior pole of the tonsil behind the posterior medullary velum (Fig 1). This segment of the artery has been designated the posterior medullary segment and corresponds to the medullary segment described by GREITZ & SJÖGREN (1963).

At vertebral angiography, this ascending part of the cranial loop is usually convex toward the midline in the half axial projection. The convexity is the result



Fig 5 Metastatic carcinoma of the left cerebellar hemisphere a) The choroid arch is depressed and flattened ( $\rightarrow$ ) The vermian branches are stretched and displaced posteriorly and inferiorly ( $\leftarrow$ ) diminishing the distance between them and the hemispheric branches ( $\rightarrow$ ) One caudal loop ( $\rightarrow$ ) dips below the level of the foramen magnum b) The low caudal loop ( $\rightarrow$ ) The hemispheric branches on the left are stretched and pulled down over the edge of the foramen magnum ( $\rightarrow$ ) indicating tonsillar herniation

of medial bulging of the tonsil into the vallecula. Occasionally, however, the PICA courses lateral to the tonsil and therefore has a normal, lateral convex curve (Fig 6). This appearance may simulate fourth ventricular dilatation or an intraventricular tumor in the fourth ventricle (Fig 7).

**Choroid arch** The PICA continues in a posterior course over the superior pole of the tonsil as the supratonsillar segment. This segment, also known as the cranial loop or choroid arch, sends out twigs to the anterior portion of the tonsil and to the choroid plexus of the fourth ventricle (Fig 1). The height of the anterior limb of the cranial loop varies less than does the location of the caudal loop (Wolf et al 1962). The apex of the cranial loop does not usually ascend as far superiorly as the fastigium of the fourth ventricle. In most instances it reaches up to the posterior medullary velum or lies 5 to 10 mm below it and sends small branches superiorly to supply the choroid plexus. The course of the supratonsillar segment, however, is variable. It may pass over the top of the tonsillar pole or course along the medial surface of the tonsil at various distances below the superior pole (Huang & Wolf 1969) (Fig 8a). In these instances a small branch usually arises from the apex of the transverse mid tonsillar segment. It then courses anteriorly and superiorly to reach the choroid plexus of



Fig 4 Same specimen as in fig 3 a) Ap view of injected specimen. Normal undulating appearance of the hemispheric branches along the undersurface of the cerebellum ( $\rightarrow$ ) b) Ap projection of vertebral angiogram. The hemispheric branches are separated from other arteries and are easily identified in this view ( $\rightarrow$ )

tonsillohemispheric branch descends along the posterior margin of the medial aspect of the tonsil. It divides into tonsillar branches that have a variable course and hemispheric branches that extend downward and posterolaterally. The hemispheric branches supply the undersurface of the hemispheres as far as its lateral border and anastomose with the anterior inferior cerebellar artery and superior cerebellar artery. The site of branching of the tonsillohemispheric stem into tonsillar and hemispheric branches varies considerably (Fig 3). Because of their common origin, however, any downward displacement of the tonsil simultaneously stretches and pulls the hemispheric branches inferiorly. The hemispheric branches are best demonstrated in the ap projection (Fig 4). As the tonsils herniate they pull and stretch the adjacent hemispheric branches carrying them down over the edge of the foramen magnum. In the lateral projection straightening may be seen of the hemispheric branches, but the stretching down over the edge of the foramen magnum is often obscured by the low caudal loop itself (Fig 5 a). In the ap projection (central ray  $5^\circ$  cephalad to orbitomeatal line) viewed tangentially at the foramen magnum these stretched hemispheric branches are easily seen (Fig 5 b).

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At vertebral angiography, this ascending part of the cranial loop is usually convex toward the midline in the half axial projection. The convexity is the result



Fig 8 a) Sagittal section of pons, medulla and cerebellum. The left PICA divides into tonillohemispheric (→) and vermis (←) branches at the mid tonsillar pole (3). The apex of the choroid loop or choroid point is not the point indicated by (→) (cf b). Choroid plexus in fourth ventricle (1); nodulus of vermis (2). b) Same specimen with nodulus and uvula of vermis removed to demonstrate a small branch (→) coursing anteriorly and superiorly to reach the choroid plexus of the fourth ventricle. Mid pole of tonsil (3).

choroid plexus it self may be identified as a tiny accumulation of contrast medium (Fig 9 c). The cranial loop may also pursue a tortuous transverse course laterally around the pole instead of the more common medial course.

In lateral films magnified 2:1 the apex of the choroid loop could be measured in 60 of 100 normal vertebral angiograms (Fig 10). Twining's line served as a reference point and a line (CD) was drawn from its midpoint to the posterior margin of the foramen magnum. A point along this line, 2.5 cm below Twining's point was designated the choroid point (CP). Measurements were made from the choroid point (CP) to the apex of the choroid arch in the 60 angiograms. These measurements were then plotted as x and y coordinates from the choroid point. The values obtained were quite variable, but assumed a radial distribution around the CP (Fig 11). The average distance from the CP to the apex of the cranial loop was 7 mm in any direction. However the range of measurements varied from 0 to 13 mm (Two cases measuring 17 mm and 18 mm respectively were also considered to be normal). Since this measurement was obtained on 2:1 magnification films, it should be corrected accordingly for nonmagnified lateral views. In the half axial projection the apex of the choroid arch or CP averaged a distance of 5 mm from the midline (Figs 12 and 13).

Despite the variation in the site of the apex of the cranial loop, the measured choroid point has a constant anatomic position. The location of the choroid point, therefore represents a helpful reference point in the diagnosis of posterior fossa masses. With minimal anterior posterior or superior inferior displacement of the CP its measurement in the lateral projection is particularly helpful. Ordinarily



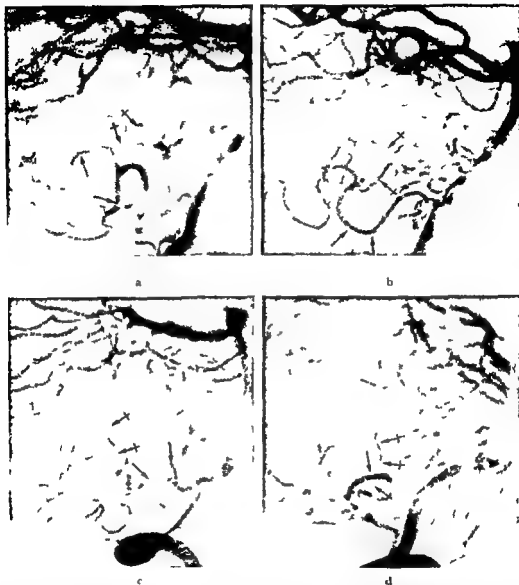


Fig. 1. Variations in the appearance of the choroid arch of the PICA as seen at angiography. a) This variant of the PICA is similar to that in the specimen of fig. 8. The PICA bifurcates at about the mid pole of the tonsil ( $\rightarrow$ ). A small branch courses anteriorly and superiorly to supply the choroid plexus of the fourth ventricle ( $\leftrightarrow$ ). b) Normal appearing caudal loop ( $\rightarrow$ ). A smaller branch supplies the choroid plexus ( $\leftrightarrow$ ). c) The PICA probably traverses the midline in this case ( $\rightarrow$ ). Small branches feed the choroid plexus ( $\leftrightarrow$ ). d) A small accumulation of contrast medium. The PICA arises below the foramen magnum and the cephalic loop appears depressed ( $\rightarrow$ ). A small branch courses superiorly to the choroid plexus ( $\leftrightarrow$ ).

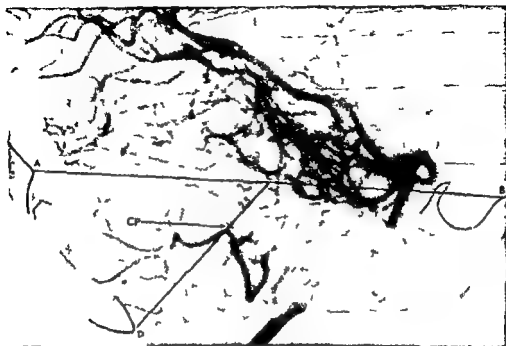


Fig 10 Measurement of choroid point on lateral film magnified 2.1



Fig 11 Radial distribution of measured choroid points around the cerebellum

Fig 12 Choroid point (→) in half axial projection

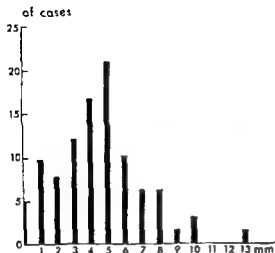


Fig 13 Measurement of choroid point from the midline in a p projection mean 5 mm



Fig 14 Pontine glioma a The posterior medullary segment ( $\rightarrow$ ) is displaced posteriorly. The choroid point  $\times$  is also posteriorly displaced when compared with the measured choroid point  $\times$  b Posterior displacement of the fourth ventricle and aqueduct by an enlarged brainstem

namely this displacement might not be readily apparent by looking at the roentgenogram (Fig 14). Displacement of the CP from side to side usually may be easily appreciated in the half axial projection even without measurements (Fig 15).

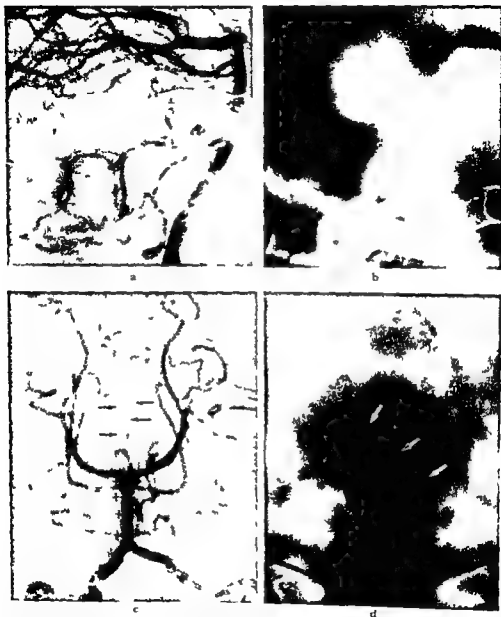


Fig 15 Recurrent cerebellar astrocytoma a) and b) No abnormality is evident in the lateral projection at angiography or at encephalography c) The posterior medullary segment of the right PICA is displaced to the left ( $\leftrightarrow$ ) indicating displacement of the right tonsil The choroid point on the right ( $\leftrightarrow$ ) is displaced slightly to the left The vermis branches ( $\rightarrow$ ) are not displaced d) At encephalography tumor is seen to encroach upon the right side of the fourth ventricle

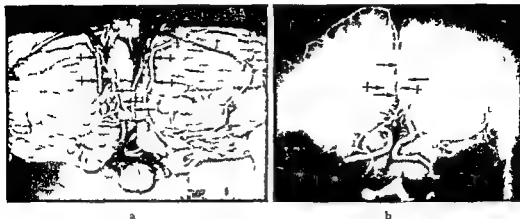


Fig 16 a) View of cerebellum from behind. The hemispheres are separated to better demonstrate the vermis branches ( $\rightarrow$ ). The medial hemispheric branches ( $\rightarrow$ ) lie 1.0 to 1.5 mm posterior to the vermis and with the hemispheres in normal position these branches would approximate each other in the midline (cf. b). Pyramid of vermis ( $\times$ ). b) Roentgenogram of specimen in (a). The medial hemispheric branches ( $\rightarrow$ ) simulate vermis branches. Identification of the smaller vermis branches is more difficult ( $\rightarrow$ ).

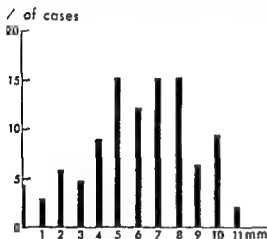


Fig 17 Measurement of vermis branch from the midline in the a.p. projection: mean 6 mm.

**Vermis branches.** The course of the vermis branches is on the lower aspect of the inferior vermis in the sulcus vallecula, between the inferior vermis and the cerebellar hemisphere. The vermis branch often forms a convex loop inferiorly and laterally. In the region of the pyramid this loop is exaggerated because the pyramid is a wider and thicker structure than the other parts of the inferior vermis (WOLF et coll. 1962).

Vermis branches are frequently multiple (MARGOLIS & NEWTON 1971, 1972).

(Fig 16 a) The total number may vary from 2 to 5 and the distance between the most lateral branches measured up to 15 mm on specimens (MARGOLIS & NEWSON)

At angiography the vermis branches often appear to have a slightly lateral convex course in the half axial projection and a posteriorly convex course in the lateral view. Since the inferior vermis does not extend as far posteriorly as the cerebellar hemispheres the vermis branches are some distance from the midline as seen in the lateral projection. In the half axial view, the most lateral vermis branch measured an average of 6 mm from the midline at the apex of its lateral convexity (Fig 17). Because of the multiplicity of vermis branches judgment as to deviation in the half axial projection may be difficult. Since a single vermis branch 1 cm from the midline still falls within the normal range the difficulty becomes even greater. Also a medial hemispheric branch may be confused with a vermis branch (Fig 16).

## SUMMARY

The more common variations in the course of the PICA that may simulate pathologic displacement are described. The caudal loop frequently extends below the foramen magnum in the normal. The diagnosis of tonsillar herniation depends on recognition of displaced tonsillar or hemispheric branches. The PICA may pursue a course lateral to the tonsil a variation that should not be mistaken for dilatation of or a tumor within the fourth ventricle. The choroid point is closely related to the posterior medullary velum. A measurement for this point is described. A false localization of the choroid point may result when the supratonsillar segment crosses the mid tonsil. Recognition of the small branches that extend to the choroid plexus of the fourth ventricle prevents this false impression. The vermis branches are often multiple and may course up to 1 cm from the midline.

## ZUSAMMENFASSUNG

Die mehr gewöhnlichen Abweichungen im Verlaufe der Arteria cerebellaris post. inf. die pathologische Verdrängungen vortauschen mögen werden beschrieben. Die caudale Krümmung erstreckt sich häufig beim Normalen unter das Foramen magnum. Die Diagnose einer tonsillären Hernienbildung hängt von der Feststellung eines abweichenden Verlaufes der tonsillären oder hemisphärischen Äste ab. Die Arteria cerebellaris post. inf. kann einem Verlauf lateral von den Tonsillen weiterverfolgen — eine Abweichung die nicht mit einer Dilatation oder einem Tumor innerhalb des vierten Ventrikels verwechselt werden sollte. Der choroidale Punkt steht in enger Verbindung zum Velum medullare posterius. Eine Messung dieses Punktes wird beschrieben. Eine falsche Lokalisation des choroidalen Punktes kann sich ergeben wenn das supratonsilläre Segment die Tonsille in der Mitte kreuzt. Die Feststellung der kleinen Äste die sich bis zum Plexus choroideus des vierten Ventrikels erstrecken vermeiden diesen falschen Eindruck. Die Äste der Vermis sind oft multipel und können sich bis zu einem cm von der Mittellinie erstrecken.

## RÉSUMÉ

Les auteurs décrivent les variations les plus fréquentes du trajet de l'artère cérébelleuse postérieure et inférieure qui peuvent simuler un déplacement pathologique. La boucle inférieure de cette artère s'étend souvent au dessous du trou occipital chez le sujet normal. Le diagnostic de hernie des amygdales repose sur la constatation du déplacement de branches amygdaliennes ou hémisphériques. L'artère cérébelleuse postérieure et inférieure peut avoir un trajet externe par rapport à l'amygdale, variation qu'il ne faudrait pas prendre pour une dilatation ou tumeur du quatrième ventricule. Le point choroidien est en rapport étroit avec la toile choroidienne postérieure. Les auteurs décrivent un procédé de mesure pour situer ce point. On peut commettre une erreur de localisation du point choroidien quand le segment sus-amygdalien croise la partie moyenne de l'amygdale. L'identification des petites branches qui s'étendent jusqu'au plexus choroïde du quatrième ventricule évite cette fausse localisation. Les branches vermiennes sont souvent multiples et peuvent s'éloigner de 1 cm de la ligne médiane.

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## OCCLUSION SEGMENTAIRE DES TRONCS SUPRA AORTIQUES PAR ARTERITE GIGANTO CELLULAIRE

Etude angiographique

par

J. M. MERCADER, A. RODRIGUEZ ARIAS Jr et J. SOLÉ LLENAS

L'occlusion des artères cervicales afférentes de l'encéphale constitue habituellement le stade final d'une lésion vasculaire athéro thrombotique.

L'étiologie occlusive artérielle est par contre moins observée bien qu'il faille en tenir compte surtout chez les adultes jeunes spécialement au niveau des troncs supra aortiques.

A côté des artérites syphilitiques infectieuses des maladies du collagène etc il existe l'artérite des cellules géantes qui constitue une entité pathologique définie.

Dans cette communication nous présentons quelques cas d'occlusions vasculaires localisées aux troncs supra aortiques d'étiologie artérielle géantocellulaire chez des patients atteints d'un syndrome d'ischémie cérébrale et qui ont été tous étudiés par angiographie opérée et vérifiés par pathologie.

### Observations

**Cas 1** Femme de 34 ans qui a été atteinte à l'âge de 12 ans d'un rhumatisme poly articulaire et de chorée mineure. À l'âge de 20 ans au cours d'un épisode de rhumatisme la malade souffrit de crises transitoires de vertige et perte de conscience. Le tout disparut



## RÉSUMÉ

Les auteurs décrivent les variations les plus fréquentes du trajet de l'artère cérébelleuse postérieure et inférieure qui peuvent simuler un déplacement pathologique. La boucle inférieure de cette artère s'étend souvent au dessous du trou occipital chez le sujet normal. Le diagnostic de hernie des amygdales repose sur la constatation du déplacement de branches amygdaliennes ou hémisphériques. L'artère cérébelleuse postérieure et inférieure peut avoir un trajet externe par rapport à l'amygdale, variation qu'il ne faudrait pas prendre pour une dilatation ou tumeur du quatrième ventricule. Le point choroidien est en rapport étroit avec la toile choroidienne postérieure. Les auteurs décrivent un procédé de mesure pour situer ce point. On peut commettre une erreur de localisation du point choroidien quand le segment sus-amygdalien croise la partie moyenne de l'amygdale. L'identification des petites branches qui s'étendent jusqu'au plexus choroïde du quatrième ventricule évite cette fausse localisation. Les branches vermiennes sont souvent multiples et peuvent s'éloigner de 1 cm de la ligne médiane.

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Fig 3

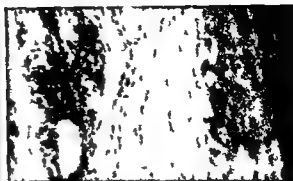


Fig 4a



Fig 4b

Fig 3 Angiographie carotidienne droite Absence de lésions sténosantes sur la carotide primitive et la carotide externe Exclusion totale de la carotide interne

Fig 4 a) Adventice avec vasa vasorum dilatés et importante infiltration inflammatoire  
b) Tunique moyenne inflammatoire Tunique interne avec restes de thrombus

**Cas 2** Homme de 35 ans qui dans ses antécédents personnels présente plusieurs poussées de rhumatisme polyarticulaire aigu. La maladie actuelle commence par une perte brusque de force et un engourdissement des deux membres du côté gauche. À l'exploration on constate une hémiplegie de l'hémicorps gauche à prédominance brachio faciale sans autre signe neurologique. L'examen électroencéphalographique montre un foyer irritatif temporo-occipital droit de situation profonde. Les analyses de laboratoire n'indiquent aucune altération. L'angiographie carotidienne droite par ponction directe montre l'absence de lésions sténosantes de la carotide primitive et de la carotide externe (Fig 3). L'absence de moignon fait penser à une aplasie de l'artère carotide interne. On ne visualise pas de circulation collatérale anastomotique à travers les branches de la carotide externe. L'angiographie de la carotide gauche montre l'absence de lésions dans la région cervicale. On opacifie tout le territoire vasculaire de l'hémisphère gauche ainsi que du droit à travers l'artère communicante antérieure. À l'intervention chirurgicale on découvre un cordon fibreux au lieu de l'artère carotide interne droite, cela depuis son origine. L'arteriotomie montre des parois artérielles très épaissies qui occupent presque toute la lumière artérielle pleine de thrombus. Anatomie pathologique. L'adventice de l'artère offre une dilatation



Fig 5



Fig 6

Fig 5 Angiographie aorta cervicale par cathétérisme de l'artère femorale. Absence complète de l'artère carotide primitive droite depuis son origine. Opacification normale du tronc brachiocephalique de la sous-clavière et de l'artère vertébrale du même côté.

Fig 6 Meso-endarterite giganto-cellulaire. Tuniques interne et moyenne avec cellules géantes.

importante des vaisseaux. Fig 4 a) La tunique médiane est discontinue, élongée et très réfringente. Prolifération de l'endartère avec présence de cellules géantes avec tentatives de recanalisation. Traînées inflammatoires du type cellules rondes de localisation prédominante dans l'adventice et foyers multiples de calcinose (Fig 4 b). Le diagnostic histopathologique a été artérite giganto-cellulaire.

**Cas 3.** Homme de 39 ans avec antécédents de rhumatisme polyarticulaire depuis la jeunesse. La maladie actuelle commence il y a deux mois par une perte de force progressive du bras et de la jambe gauches. L'exploration clinique confirme l'existence d'une hémiparésie gauche affectant la face du même côté. L'examen électroencéphalographique montre l'existence d'un foyer irritatif temporo-pariétal droit de situation profonde. Les données de laboratoire étaient dans les limites normales. Sur l'angiographie aorto cervicale par cathétérisme de la femorale on observe l'absence complète de l'artère carotide primitive droite depuis son origine. Opacification normale du tronc brachiocephalique, artère sous-clavière et artère vertébrale du même côté (Fig 5). La vérification chirurgicale montre la transformation de la carotide primitive droite en un cordon fibreux sur tout son parcours. À l'examen histologique l'adventice apparaît légèrement cellulaire, très riche en collagène, filot hyalin, mal délimitée de la couche élastique dans laquelle il apparaît une limitante très réfringente. Limita est épaissie, quelque peu mamelonnée et la lumière vasculaire fortement réduite. L'intense inflammation de caractère mononucléaire retient notre attention ainsi que la présence de multiples cellules géantes de localisation méso-arterielle et principalement dans les zones de ruptures de la limitante (Fig 6). Il s'agit donc d'une méso-endarterite giganto-cellulaire.

### Commentaires

Nous présentons trois cas de lésions occlusives artérielles d'étiologie inflammatoire géantocellulaire tous se présentant dans la quatrième décade de la vie et avec des antécédents de rhumatisme polyartculaire dans leur histoire clinique.

Dans deux de ces cas la lésion siège à l'origine du vaisseau. Elle se traduit à l'angiographie par une absence complète d'opacification de l'artère sans moignon contrairement à ce qui arrive dans les occlusions par athéro-thrombose. L'image rappelle au premier abord celle d'une aplasie artérielle.

Ces trois cas à l'encontre des lésions artériosclérotiques ■ caractérisent par la sélectivité et la limitation des lésions à une seule artère ce qui facilite une intervention chirurgicale éventuelle.

### RÉSUMÉ

Une étiologie rarement observée d'occlusion des artères cervicales extracrâniennes est l'arterite géantocellulaire non spécifique. Nous présentons trois patients atteints d'un syndrome d'ischémie cérébrale. Nous décrivons les signes angiographiques dont on peut tenir compte pour le diagnostic différentiel avec d'autres lésions occlusives et nous commentons les conclusions obtenues par l'étude de ces cas compte tenu de l'âge des antécédents pathologiques de la symptomatologie clinique et des données histopathologiques obtenues.

### SUMMARY

One of the rarer causes of occlusion of the extracranial cervical arteries is nonspecific giant cell arteritis. Three such cases with resulting cerebral ischaemia are reported. The angiographic signs distinguishing this from other occlusive conditions are described. Based on these observations and with due regard to the age of the patient, the previous history, clinical signs and histologic findings a firm diagnosis should be possible.

### ZUSAMMENFASSUNG

Eine der seltenen Ursachen, die zum Verschluss der extracraniellen Halsarterien führen, ist die unspezifische Riesenzellenarteritis. Über drei einschlägige Fälle, die an ischaemischen Gehirnsymptomen litten, wird berichtet. Die für die Differentialdiagnose wichtigen angiographischen Zeichen, die es ermöglichen eine richtige Diagnose zu stellen, werden herausgestellt. Falls diese Zeichen gewürdigt werden und das Alter der Patienten, die Vorgeschichte, die klinischen Symptome und die Histologie in Betracht gezogen werden, sollte eine sichere Diagnose möglich sein.

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## AORTOCERVICAL AND CEREBRAL ANGIOGRAPHY IN EXTRACRANIAL CEREBROVASCULAR DISEASE

by

R OBERSON

The management of cerebral vascular accidents is always based on a correct angiographic diagnosis and it is of course the neuroradiologist who must determine whether the stroke is to be classed as a neurosurgical or vascular surgical entity.

*Material* This consists of 200 aortocervical angiographies performed in patients with cervical arterial or cerebrovascular conditions compared with cerebral angiographies. No statistical investigation has been undertaken because both the indications and technical procedures have been continuously modified during the sixteen month period.

*Method* Cerebral angiography is first performed by low direct puncture of the common carotid artery on the side probably involved. If aortography is performed first risks of clinical worsening are higher because the patient has already had a minimum dose of 100 ml of contrast medium injected into the aorta before the selective carotid angiography of the affected area. Aortography will determine only the presence or not of any extracranial pathology. The carotid artery is perforated well below the bifurcation and lateral stereoscopic films are obtained with an AOF film changer (Elena Schonander) following the injection of 3 to 5 ml metrizoate methylglucamine (280 mg I/ml). The head is

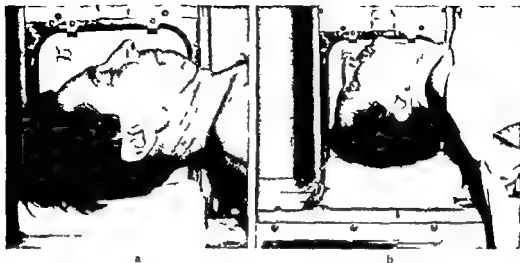


Fig 1 a) Normal head position for lateral cerebral angiography b) Hanging down position of the head for demonstration of the carotid bifurcation in patients over 40 (this position in younger patients often brings the cannula into the external carotid artery)

tilted back, so as the carotid bifurcation and the cerebral arteries are included (Fig 1)

Stereoscopic views are helpful in demonstrating small atheromatous lesions not so much for stereoscopic purposes but mainly for supplying simultaneous slightly oblique views of the carotid bifurcation. MADDISON & MOORE and WOOD & CORRELL stated that ulceration is best seen in the lateral projection. The common carotid arteries are often the least damaged vessels in cerebral accidents. A second injection of 3 to 5 ml metrizoate methylglucamine may be made for a posterior films if necessary. An aortocervical angiography may be performed later in the chronic stage.

Aortography is followed by indirect selective catheterization in transient ischemic and stroke in evolution attacks. Diffuse degenerative extra and intracranial vascular pathology probably exists in such cases.

Transient ischemic and stroke in evolution attacks demand that selective carotid and vertebral angiographies are performed after the aortocervical examination. Angiography in the ischemic attacks may be programmed but must be made without delay in cases of stroke.

### Discussion

The lateral cranial view must include the carotid bifurcation in patients over 40 years old submitted to angiography. This is achieved by tilting the head so



Fig 2 a) Normal head position (cf Fig 1 a) Only external carotid artery branches filled b) Hanging head position of the head (cf Fig 1 b) in the same patient with the tip of the cannula (→) the carotid bifurcation and the occlusion of the internal carotid artery (→) demonstrated The orbito-mental line (white line) varies in both positions c) Vertebral angiography reveals the extent of the thrombus by the reflux of the contrast medium into the carotid siphon (cavernous part) (→) a defect (→) at the distal end of the siphon indicates that a small embolus occludes the origin of the middle cerebral artery

as the fronto-occipital diameter lies across the 24 cm  $\times$  30 cm film changer (the largest head diameter in the shortest film dimension) (Fig 1) Certain small but important lesions are demonstrated only in this position. The necessity of including the carotid bifurcation has been stressed by NEWTON & COLCH. The needle point must be included in the films if technical errors at the site of injection are to be recognized. With the equipment in current use however there is a gap of about 2 cm between the edge of the cassette holder and the edge of the film. The lateral projection is preferable since in it the proximal parts of the internal and external carotid arteries are usually not superimposed. It would appear that the hanging of the head prevents any difficulties. The present author has failed to demonstrate the bifurcation only in 5 per cent of cases.

Extracranial internal carotid artery occlusion demands thorough examination of the circle of Willis by bilateral angiography of both carotid and vertebral arteries. It is of prime necessity to recognize the extent of the obstruction whether it is involving part of the carotid siphon or is localized at the bifurcation only (Fig 2). The vascular conditions in the neighbourhood of the region of a tumour or aneurysm are also most important. Other sites of vascular pathology and the collateral circulation have to be demonstrated before operation.



## Acknowledgements

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## SUMMARY

A material of 200 patients with cerebral vascular accidents examined by aortocervical angiography and selective carotid and vertebral angiographies is discussed. Positioning for the procedures which is most important is described.

## ZUSAMMENFASSUNG

Ein Material von 200 Patienten mit cerebrovaskulären Läsionen, die mittels aortocervikaler Angiographie und selektiver Angiographie der A. carotis und der A. vertebralis untersucht wurden, wird diskutiert. Die richtige Lagerung der Patienten, die sehr wichtig ist, wird beschrieben.

## RÉSUMÉ

L'auteur analyse une série de 200 malades atteints de lésions cerebrovasculaires examinés par angiographie aortocervicale et d'angiographies sélectives carotidienne et vertébrale. Il décrit la mise en place du malade pour ces examens, ce qui est très important.

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## DIAGNOSTISCHE SCHWIERIGKEITEN ANGIOGRAPHISCHER BEFUNDE BEI ATYPISCH LOKALISIERTEN ANEURYSMEN

VON

H OTTO W BETTAG E LOHR und W GROTE

Die Behandlung cerebraler Aneurysmen gehört heute mit zu den dankbarsten und erfolgreichsten Aufgaben der Neurochirurgie. Voraussetzung ist eine exakte Lokalisationsbestimmung, die ausschließlich mit Hilfe der Angiographie getroffen werden kann.

Am häufigsten findet man die sackförmigen Aneurysmen am vorderen Teil des Circulus Willisii, des weiteren an den Stämmen und Teilungsstellen der großen Hirngefäße. Selten sind Aneurysmen in den peripheren Stromgebieten der Hirnarterien. Über Aneurysmen peripherer Mediaäste, von denen hier berichtet werden soll, existieren in der Literatur nur wenige Mitteilungen. Einzelne Fälle werden von LINDGREN (1954), JANNY et coll (1962), KIMBELL & LLEWELLYN (1960) und SINGH (1968) mitgeteilt. LOCKSLEY findet in der amerikanischen Siebenjahresstudie von 1958 bis 1965 über Aneurysmen und Subarachnoidalblutungen unter 2695 einzelnen rupturierten und nicht rupturierten Aneurysmen 37 (1,4%), die distal der großen Äste der A. cerebri media gelegen sind. Betrachtet man die Zahlen von AF BJÖRKESTEN (1967) der 2177 Patienten mit Subarachnoidalblutungen angiographisch untersuchte und dabei 1601 Aneurysmen entdeckte, so wird deutlich, daß unter peripher gelegenen Aneurysmen diejenigen der A. cerebri media am seltensten vorkom-

men. Er fand 76 Aneurysmen an der A. pericallosa, A. callosomarginalis und A. frontopolaris, jedoch nur zwei Fälle an kleinen Mediaasten.

Die Angaben treffen für die angeborenen (Forbushen) Aneurysmen zu. Diejenigen embolisch mykotischer Genese dagegen sind vorwiegend an der A. cerebri media gelegen (STENGEL & WOLFERTH 1923, DANDY 1944, RATHMELL et coll. 1952) wobei eine Bevorzugung der peripheren Gefäßanteile festzustellen ist (ROACH & DRAKE 1965, PETIT, DUTAILLIS & PITTMAN 1955). Es muß jedoch betont werden, daß klinisch und angiographisch diagnostizierte Fälle von mykotischen Aneurysmen ebenfalls eine große Seltenheit darstellen (KING 1960, AGNOLI & BETTAG 1971).

Die Therapie peripher gelegener Aneurysmen gestaltet sich insofern einfacher, als man zumeist das zuführende Gefäß unterbinden kann, ohne daß grobere neurologische Ausfallerscheinungen zu erwarten sind. Komplizierter wird die Situation, wenn es zur Ruptur gekommen ist. Die daraus resultierende Blutung, die häufig nach intracerebral erfolgt (WEICKMANN 1969), hat nicht minder verheerende Folgen als bei Aneurysmen anderer Lokalisation (ROACH & DRAKE 1965). McHISOCK & WALSH (1956) fanden bei 42 Aneurysmen der A. cerebri media in 10 Fällen ein intracerebrales Hamatom. Die Mortalität bei chirurgisch behandelten Fällen betrug über 50 %. In einem Obduktionsmaterial von 33 Fällen mit Aneurysma der A. cerebri media fand CROMPTON (1962) in 23 Fällen eine intracerebrale Blutung.

Im folgenden soll über drei eigene Beobachtungen berichtet werden, bei denen angiographisch ein Aneurysma eines peripheren Mediaastes nachgewiesen wurde und aufgrund der Gefäßverlagerung eine raumfordernde Blutung angenommen werden mußte.

**Fall 1.** Die 35-jährige Patientin wurde uns unter der Verdachtsdiagnose Hirnblutung überwiesen. Aus der Vorgeschichte war bekannt, daß sie mit 16 Jahren einen akuten Gelenkrheumatismus durchmachte. Seither war bei ihr ein Vitium cordis bekannt. Im letzten Jahr verschlechterte sich ihr Allgemeinzustand ständig. Sie litt unter Herzschmerzen, Müdigkeit und Inappetenz. Es kam zu einer Gewichtsabnahme von 10 kg. Gut 4 Wochen vor der Krankenhauseinweisung stellten sich Schwellungen, Rötungen und Schmerzen in Sprunggelenken und Kniegelenken ein. Es wurden eine deutliche Leber- und Milzschwellung, eine Anaemie, Erhöhung der BSG und eine Leukocytose festgestellt. Am Abend vor der Verlegung in unsere Klinik traten plötzlich ein heftiger rechtsseitiger Kopfschmerz, anhaltendes Erbrechen und nachfolgend eine tiefe Bewußtlosigkeit auf.

Bei der Aufnahme hier war die Patientin tief komatös, sie reagierte eben auf Schmerzreize. Im Befund waren Nackensteifigkeit sowie eine rechtsseitige Mydriasis. Am Augenhintergrund sah man beiderseits eine Papillenunschärfe und Netzhautblutungen. Das Babinski'sche Zeichen ließ sich beiderseits auslösen. Im EEG fanden sich mittelschwere Allgemeinveränderungen mit Zeichen eines Hirndrucks und einem Thetafokus rechts temporal. Bei der Lumbalpunktion konnte xanthochromer Liquor gewonnen werden. Die rechtsseitige Carotisangiographie (Abb. 1) zeigte eine deutliche Verdrängung der A. pericallosa nach links.

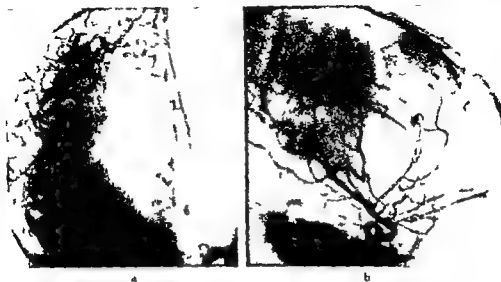


Abb 1 Fall 1 Rechtseitige Carotisangiographie Kirschkeimgroßes Aneurysma einer frontalen Operculararterie a) a.p. Projektion Deutliche Verdrängung der A. pericallosa zur Gegenseite b) Seitliche Projektion Herabdrängung der A. cerebri media als Zeichen einer intracerebralen raumfordernden Blutung

so wie eine Herabdrängung der A. cerebri media. Außerdem stellte sich im Bereich einer frontalen Operculararterie ein etwa kirschkeimgroßes Aneurysma dar.

Bei der sofort durchgeführten rechts frontalen Craniotomie stieß man im subcorticalen Marklager auf eine gut kirschkeimgroße Blutung, die aus koagulierten Blutmassen bestand. Nach Ausräumung des Hämatoms konnte im Bereich der Rinde ein kleines Konvolut, welches dem röntgenologisch dargestellten Aneurysma entsprach, unterbunden und exsurpiert werden. Postoperativ erholte sich die Patientin vorübergehend, sie reagierte auf Anruf und bot keine neurologischen Ausfallserscheinungen. Am 4. Tage kam sie jedoch plötzlich unter dem Bild eines Herz- und Kreislaufversagens ad Exitum. Bei der Sektion fanden sich am Herzen deutliche Zeichen einer abgetriebenen Endocarditis mit frischen entzündlichen Veränderungen an den Klappen. Außerdem bestanden ein ausgedehnter Infarkt der rechten Niere, ein Verfettungsgrad der Leber und eine Milzvergrößerung. Bei der histologischen Untersuchung des bei der Operation entnommenen Präparates wurde ein mykotisches Aneurysma diagnostiziert.

Fall 2 Der 67-jährige Patient war uns seit Anfang 1969 bekannt, als er wegen radikularer Schmerzen bei uns behandelt wurde. Im November desselben Jahres traten bei ihm erstmals linksseitige Jackson-Anfälle auf. 14 Tage danach bemerkte der Patient eine leichte Lahmung der linksseitigen Extremitäten. Die Anfallshäufigkeit sowie die Lähmung nahmen im weiteren Verlauf zu. Der Patient wurde deshalb erneut bei uns stationär aufgenommen. Zu diesem Zeitpunkt bestand eine ausgeprägte linksseitige spastische Hemiparese. Eine Stauungsrippe war nicht nachweisbar. Das EEG zeigte eine rechts fronto-temporale Alpha-Aktivierung. Bei der rechtseitigen Carotisangiographie (Abb. 2) zeigte ein etwa kirschkeimgroßes



Abb 2 Fall 2 Rechtsseitige Brachialangiographie. Linsengroßes Aneurysma einer parietalen Operculararterie a) a.p. Projektion A. pericallosa nach links über die Mittellinie verschoben b) Seitliche Projektion Mediagruppe leicht angehoben

**Aneurysma einer Operculararterie.** Die A. pericallosa war über die Mittellinie nach links verschoben, die Mediagruppe leicht angehoben. Unter dem Verdacht einer raumfordernden Blutung aus dem Aneurysma craniotomierten wir den Patienten. Nach Eröffnen der Dura zeigte sich die Hirnoberfläche unauffällig und bei der Palpation konnte keine Konsistenzänderung festgestellt werden. Auch die Punktion war negativ, es fanden sich keine Anzeichen für ein intracerebrales Hamatom. Das Aneurysma, das am Fuße der Präcentralregion gelegen war, wurde proximal und distal verklippt und exstirpiert. Postoperativ erholte sich der Patient nur sehr langsam; eine Kontrollangiographie konnte ihm wegen des schlechten Allgemeinzustandes nicht zugemutet werden. Vier Monate nach der Operation kam er ad Exitum. Bei der Sektion fand sich als Todesursache ein Bronchialcarcinom, welches trotz mehrfacher Röntgenaufnahmen des Thorax vorher nicht bekannt gewesen war. Die Hirnsektion ergab regelrechte Verhältnisse; insbesondere ließen sich keine raumfordernden Blutungen nachweisen.

**Fall 3.** Der 40-jährige Patient war vorher nie ernsthaft erkrankt. Er litt jedoch seit 10 Jahren häufig an Kopfschmerzen über beiden Schläfen. Sechs Monate vor der stationären Aufnahme bemerkte er plötzlich starke Kopf- und Augenschmerzen, die bereits am nächsten Tage nachließen; jedoch stellte er beim Blick nach rechts Doppelbilder fest. In der Folgezeit traten auch Übelkeit, Erbrechen und kurz vor der Aufnahme linksseitige Jackson-Anfälle auf. Bei der Einweisung bestand eine leichte schlaffe Hemiparese links sowie eine rechtsseitige Abducensparese. Im EFG konnte eine rechts centro-parietale Alphaänderung als Seitenhinweis gewertet werden. Im rechtsseitigen Carotidangiogramm (Abb. 3) kam ein Aneurysma von etwa 1,5 mm × 3 mm Größe im Bereich einer parietalen Operculararterie

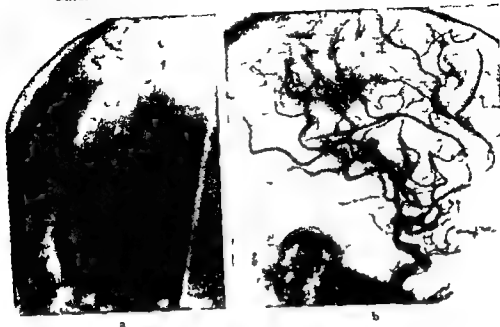


Abb 3 Fall 3 Rechtseitige Carotisangiographie kirschkerngroßes Aneurysma einer parietalen Operculararterie a) a.p. Projektion A. pericallosa mittelständig b) Seitliche Projektion. Bogenförmiger Verlauf der hochgedrängten A. cerebri media

zur Darstellung. Die A. cerebri media war deutlich nach medial und hochgedrängt, während die A. pericallosa mittelständig war.

Unter dem Verdacht einer raumfordernden Blutung erfolgte auch hier die Operation. Dabei konnte man sich jedoch weder von einer subarachnoidalen noch einer intracerebralen Blutung überzeugen. Auf eine Isehrpräparation des Aneurysmas wurde verzichtet, um eine Verletzung der Zentralregion zu vermeiden. Postoperativ erholte sich der Patient sehr gut. Unter antikonvulsiver Medikation wurden keine Anfälle mehr beobachtet, und die übrigen Krankheitserscheinungen wurden rückläufig, mit Ausnahme der Abducensparese. Der Patient ist heute 1 Jahr nach der Operation wieder in seinem alten Beruf als Maschinensteiger tätig.

### Diskussion

Allen drei beschriebenen Fällen ist das Vorkommen eines Aneurysmas ungewöhnlicher Lokalisation gemeinsam mit allerdings unterschiedlicher Vorgeschichte und klinischem Verlauf. Im ersten Fall sind die typischen Merkmale einer Aneurysmaruptur mit plötzlichen heftigen Kopfschmerzen, anhaltendem Erbrechen und nachfolgender tiefer Bewußtlosigkeit zu beobachten. Die Vorgeschichte mit akutem Gelenkrheumatismus in der Jugend und den Zeichen eines erneuten Schubes eines rheumatischen Fiebers bei bereits vorgeschädigtem

Herzen geben einen Hinweis auf die Ätiologie des Aneurysmas. Durch die histologische Untersuchung konnte die mykotische Genese gesichert werden. Die angiographisch nachgewiesene Raumforderung ließ sich im Zusammenhang mit dem dargestellten Aneurysma nur als intracerebrale Blutung deuten. Bei diesem Befund blieb als einzige therapeutische Konsequenz die sofortige Craniotomie mit Aufräumung des Hämatoms und Ausschaltung des Aneurysmas. Durch die es Vorgehen ließ sich eine deutliche Besserung im Krankheitsbild erzielen. Für den weiteren ungünstigen Verlauf ist nicht zuletzt das schwere Grundleiden ausschlaggebend gewesen.

Eine andere Symptomatik und unterschiedlichen Verlauf boten die beiden anderen Patienten. Der schleichende Beginn sowie die zuletzt im Vordergrund stehenden Jackson-Anfälle und Hemiparese sprachen mehr in erster Linie für ein Aneurysm oder eine Aneurysmarruptur, wenngleich solche Verläufe nicht eher selten gefunden werden. LOSSIS et coll. (1957) fanden bei 109 Patienten mit Aneurysmen in 17% solche von paralytischen Typ und in 0,9% eine Anfall anamnese. BJORKSTEN (1958) beobachtete bei 52 Aneurysmen der Arteriae cerebri mediae 22 mit Hemiparese oder Hemiplegie und 5 Patienten mit Anfällen, wobei in einem Fall die es Symptom im Vordergrund stand. Wenn auch einem Aneurysm der Arteriae cerebri mediae kein charakteristisches Syndrom zugeordnet werden kann (LOSSIS et coll. 1957), so ist die Symptomatik in unseren beiden Fällen aufgrund der Aneurysmlokalisation durchaus erklärlich. Bei den bereits bekannten diagnostischen Schwierigkeiten von peripher gelegenen Aneurysmen, orthograd getroffene Gefäße, Überlagerung von Gefäßschlingen, die auch bei Anwendung des elektrischen Subtraktions- und Harmonisierungsverfahrens nicht beseitigt werden können, rechtfertigt der Nachweis einer solchen Gefäßneubildung allein ein operatives Vorgehen nicht. Der klinische und angiographische Hinweis auf eine raumfordernde Blutung waren ausschließlich maßgebend für unsere Indikationsstellung. Auch retrospektiv hätten wir es nicht für möglich mit Hilfe anderer Methoden wie Encephelographie und Hirnangiographie eine sichere Diagnose in allen drei Fällen zu stellen. Eine endgültige Klärung kann nur durch die Craniotomie erfolgen. Bei der angesprochenen Schwierigkeit unserer Beobachtungen hätten wir es für weise gehalten, auf die diagnostischen Schwierigkeiten hinzuweisen.

## ZUSAMMENFASSUNG

Es wird über drei Fälle von Aneurysmen der Arteriae operculares berichtet. Aufgrund des angiographischen Befundes mußte in allen Fällen der Verdacht auf die Vorliegen einer raumfordernden Blutung aus dem rupturierten Aneurysma geäußert werden, was intraoperativ jedoch nur in einem Fall bestätigt werden konnte. Auf die Schwierigkeit der Differentialdiagnose anhand des angiographischen Bildes wird hingewiesen.

## SUMMARY

Three cases of aneurysm of the opercular arteries are reported. The angiographic appearances suggested a space occupying haemorrhage from a ruptured aneurysm but as this could be confirmed in one case only the differential diagnosis in such cases must leave room for doubt.

## RÉSUMÉ

Présentation de trois cas d'anévrisme des artères operculaires. Dans tous ces cas l'angiographie devait faire soupçonner la présence d'un hématome expansif provenant de l'anévrisme rompu. Cependant cet hématome n'a pu être constaté peropératoirement que dans un cas. Les auteurs insistent sur la difficulté du diagnostic différentiel fondé sur les images angiographiques.

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## CHEMODECTOMAS OF THE HEAD AND NECK

by

ENRIQUE PALACIOS

The characteristic slow growth of chemodectomas frequently causes a delay in diagnosis and their anatomic location adjacent to critical structures, results in surgical difficulties and considerable morbidity. In our experience with 30 chemodectomas of the head and neck roentgenographic evaluation has been of great value in assessing the extent of these lesions for determining the best method of treatment.

The term 'chemodectoma' was first proposed by MULLIGAN (1950) to describe a neoplasm arising from the chemoreceptor system. Glomus tumor and non chromaffin paraganglioma are synonyms.

According to SESSIONS et coll (1959) the chemoreceptor system is composed of a number of histologically similar structures located in various parts of the body. They consist of groups of cells with sensory innervation which respond reflexly to changes in the chemical composition of the arterial blood.

The chemoreceptor bodies are named according to their location: tympanic bodies, glomus jugulare, vagal body, carotid body, laryngeal bodies and tracheal bodies.

Chemodectomas of the ciliary ganglion of the eye and in the mandible have been described by FISCHER & HAZARD (1952) and LAYTAS et coll (1954) however normal chemoreceptor tissue in these areas has not been described.

Tumors may be found arising from the chemoreceptors located in regions other than head and neck, such as in the mediastinum from the aortic pulmonary bodies as reported by LATTIS (1950) and DUNCAN & McDONALD (1951), and in the retroperitoneal and femoral areas reported by SMITANA & SCOTT (1951) and RANDALL & WALTER (1954).

Chemodectomas are slow growing and extremely variable in size ranging from a few millimeters to ten or more centimeters. They are usually encapsulated but may sometimes be intimately bound to vessels and other adjacent structures. Although the vast majority of chemodectomas are histologically benign they may invade locally or recur and infrequently metastasize. ROMANSKI (1954), ROSENWASSER (1958) and SURFELMAN & ROSENBLUTH (1959) have demonstrated such instances; however this was not found in our series.

The chemodectomas are much more common in females than in males particularly the glomus tumors located in the tympanic jugular area.

In our series the ages ranged between 29 and 70 years. Intratympanic tumors were the most common ones (18 lesions). Seven glomus jugulare tumors were encountered, 4 carotid body tumors and 1 vagal body tumor.

Glomus tumors located in the tympanic and jugular areas are regarded as the most common neoplasms involving the middle ear (25 patients in our group) and found to be extremely common in female.

The majority are unilateral but bilateral tumors are occasionally found. The latter instance was encountered in one of our patients. Simultaneous occurrence of carotid body tumor and glomus jugulare tumor was found in one of our cases as also recorded by KILMER (1917) and by SCHAEFF (1953). Other simultaneous occurrence such as carotid body tumors and vagal tumors has been recorded by MARCUS & CHAMBERLIN (1956) and carotid body tumor and chemodectoma of the orbit was recorded by LATTIS et coll. (1954). The rare features of heredo-familial tendency have been shown by DESAI & PATEL (1961) by BORDER (1967) and demonstrated by us in one patient.

*Carotid body tumors.* The best known chemoreceptor is the carotid body. It is a small mass measuring about five millimeters, highly vascularized and containing chemoreceptor cells surrounded by vascular sinusoids. Its nerve supply, according to ANDERSON & SCARFELLA (1963) and SCHLEGELTER & CHAUSIN (1966), is predominantly sensory via a branch of the glossopharyngeal nerve. Its blood supply comes from the external carotid artery.

Carotid body tumors present themselves as painless masses in the lateral aspect of the neck. Due to slow growth and the lack of or minimal symptoms it is not usually discovered until it has grown for some time. The duration of growth may range from months to many years before detection. When the lesion is large it may produce pressure on the surrounding structures causing dysphagia, hoarse



Fig 1

Fig 2

Fig 1 Highly vascularized carotid body tumor revealing extensive arterio-venous shunts contrast filling of the internal jugular vein

Fig 2 Modified submento-occipital view demonstrating enlargement of the right jugular fossa (arrows) produced by a glomus jugulare tumor

ness, cough and nausea. Despite the close relationship between the carotid sinus and the tumor, carotid sinus symptoms are remarkably rare.

Physical examination as a rule reveals only a tumor mass at the carotid bifurcation. The mass may be pulsatile and may be associated with a thrill or bruit, making its differentiation from an aneurysm difficult or impossible.

The profuse vascularity of these tumors makes carotid angiography the most valuable diagnostic tool, especially if surgery is contemplated. Angiography yields important information concerning the size of the lesion, the relationship with the carotid vessels and other adjacent structures. Precise diagnosis is established in the great majority of cases. The tumor appears to be well circumscribed. Contrast medium accumulates within a network of small irregular vessels and — not infrequently — arterio-venous shunts are demonstrated (Fig 1). The differentiation from carotid aneurysm is not difficult. Usually the carotid vessels at the bifurcation are separated from each other by the tumor mass and may appear slightly narrowed by pressure but are almost never occluded.

The cerebral blood flow from the opposite side of the lesion should be assessed. Knowledge of the patency of the collateral circulation via the circle of Willis or extracranial vessels is of extreme importance should ligation of the affected artery become necessary during the surgical procedure.



Fig 3 a) A post mortem demonstrating an extensive destruction of the right jugular fossa by a glomus jugular tumor extending into the hypotympanum (arrows). Associated inflammatory changes b) Normal left side for comparison

*Intratympanic and glomus jugulare tumors* GUNN (1941 and 1953) described this important group of chemoreceptor—the glomus jugulare complex, located in the jugular and middle ear area. These glomera are situated mainly along the structures of the glossopharyngeal nerve by the superior ganglion, in the adventitia of the bulb of the internal jugular vein along the tympanic nerve and in the tympanic plexus (tympanic bodies), located in the mucosa of the cochlear promontory. Glomera may also be found along the auricular branch of the vagus nerve sometimes as far as the facial canal.

Each ear contains from two to three glomera. They tend to be bilateral and symmetrical in location. They measure about one millimeter in diameter. Histologically they are similar to the carotid body and other chemoreceptors. They are innervated by the glossopharyngeal nerve. The blood supply is mainly derived from the external carotid artery via the tympanic branch of the ascending pharyngeal artery.

Intratympanic glomus tumors produce a variety of symptoms determined by the location, direction of spread of the tumor and stage at which the patient is seen. These tumors are usually detected when a polypoid mass breaks through the tympanic membrane or the wall of the external auditory canal. Early the lesion may be manifested by a slight bluish discoloration produced by a mass behind the tympanic membrane. The usual symptoms are: (1) progressive hearing loss, tinnitus (often pulsatile), vertigo, pain, (2) bloody discharge and secondary infection after rupture of the tympanic membrane, and (3) occasional isolated facial paralysis. A combination of otologic and neurologic symptoms may occur when the tumor extends to the petro-tympanic and jugular areas, or when the lesion penetrates the dura into the middle and posterior cranial fossae.



Fig 4



Fig 5

Fig 4 Carotid angiogram. Small glomus jugulare tumor demonstrated with the aid of subtraction technique

Fig 5 Subtraction film of a carotid angiogram in the late arterial phase demonstrating a large glomus jugulare tumor (→) Contrast filling of a large vein (→) through arterio-venous shunts

Glomus jugulare tumors may compress the jugular bulb and may extend into its lumen. If the lesion destroys the roof of the jugular fossa the middle ear or external auditory canal may be involved producing symptoms as if the tumor had arisen from the tympanic cavity. If the lesion extends posteriorly and medially into the jugular foramen, it may produce paresis of the 9th, 10th, and 11th cranial nerves. The latter occasionally may be the only clinical manifestation of the lesion as was the case in one of our patients. Further extension medially may involve the 12th cranial nerve as it passes through the hypoglossal canal. Occasionally the trigeminal and abducens nerves are affected when the lesion reaches the middle cranial fossa through the tip of the petrous bone.

Roentgenographic detection of these tumors is important since the clinical



Fig 6



Fig 7

Fig 6 Subtraction film of a selective external carotid angiogram demonstrating a small glomus jugulare tumor

Fig 7 Internal jugular phlebogram Complete obstruction of the vein at the jugular bulb (arrow) produced by a glomus jugulare tumor extending into its lumen

manifestations are frequently so vague. Initially, intratympanic tumors are manifested by soft tissue swelling within the tympanic cavity and the petro-mastoid cells. However, the commonly superimposed infection which follows the perforation of the tympanic membrane by these tumors is often indistinguishable from otitis media. As the lesion progresses bone destruction may be detected in the hypotympanic area, mastoid cells, external auditory canal and jugular fossa.

If the glomus jugulare tumor is large enough it will destroy the roof of the jugular fossa into the middle ear and adjacent structures. At this stage it is difficult to determine the site of origin. Accurate diagnosis necessitates a knowledge of numerous normal anatomic variations of the bony structures; the recognition of a large and asymmetric jugular foramen as a normal variation should not be confused with a pathologic enlargement (Fig 2). Occasionally



Fig 8 Carotid angiogram. Large vascular body tumor extending medially into the pharynx.

the tumor may extend anteriorly involving the posterior portion of the temporomandibular joint

Tomography is the most helpful method for demonstrating destructive changes involving the jugulo-tympanic and sublabrynthine areas (Fig 3 a and b)

Angiography provides excellent demonstration of the size, the extension of the vascularized lesions, and significantly aids in the differential diagnosis (Fig 4). Glomus jugulare tumors are mainly supplied by the ascending pharyngeal artery of the external carotid artery. Meningeal branches from the internal carotid and vertebral arteries may also contribute to their supply, especially if the lesion extends into the middle and posterior cranial fossae. Frequently arterio-venous shunts are demonstrated in large glomus jugulare tumors and it is easy to confuse them as an arterio-venous malformation (Fig 5).

Selective external carotid angiography with the aid of subtraction technique demonstrates lesions to a better advantage and may actually be the only method to demonstrate small tumors (Fig 6).

Intraluminal invasion of the internal jugular vein and jugular bulb by glomus tumors may readily be seen with internal jugular phlebography as described by CEJROT & LAUREN (1964), and should be considered a valuable preoperative procedure complementing the information of the other examinations (Fig 7).



*Vagal tumors* The third important site for the chemoreceptor tissue to occur has been reviewed by BURMAN (1955) and BERK (1961), and is found in the vagus nerve in its cervical portion, around the inferior ganglion or ganglion nodosum (vagal bodies) and along the rest of the cervical portion (glomus intravagale)

The usual clinical manifestation of vagal tumors is a painless mass behind the angle of the mandible which may bulge into the pharyngeal wall producing dysphagia. Since the mass lies in close relation to the carotid vessel, it may be pulsatile simulating an aneurysm. Chemoreceptor hyperactivity and involvement of the 9th, 10th, 11th and 12th cranial nerves may occur.

Angiography yields specific information, demonstrating a vascularized mass above the carotid bifurcation, below the jugular foramen, and displacing the carotid vessels laterally and posteriorly (Fig. 8).

*Laryngeal and tracheal chemodectomas* As of now we have no roentgenographic experience with such rare tumors. The few reported cases by ANDREWS (1955), ZEFMAN (1956), MCCART & KARAM (1958), HARTMAN (1960), HOLLEREE (1963) and MARTINSON (1967), have presented them chief as a subglottic mass involving the vocal cords producing laryngeal obstruction extending into the thyroid area. Conventional films and tomography of the larynx and trachea have been used in the diagnosis. There is no angiographic experience recorded; however, the profuse vascularity of these lesions should make angiography an important tool for better preoperative evaluation.

### Conclusions

Symptoms and signs indicate in the majority of cases the presence of chemodectomas. However, the roentgenographic evaluation with appropriate conventional films, tomography and angiography is necessary to confirm the diagnosis and assess the extension of the lesion.

### SUMMARY

Anatomic, clinical and roentgenographic features of chemodectomas of the head and neck have been reviewed. The experience with 30 such lesions is presented. The importance of the various roentgenographic techniques in the assessment of these lesions is stressed.

### ZUSAMMENFASSUNG

Die anatomischen, klinischen und röntgenographischen Merkmale von Chemodectomen des Kopfes und Nackens werden zusammenfassend besprochen. Die Erfahrungen von 30 derartigen Tumoren werden dargestellt. Die Bedeutung der verschiedenen röntgenographischen Techniken bei der Beurteilung dieser Schäden werden hervorgehoben.

## RÉSUMÉ

L'auteur fait une revue des caractères anatomiques cliniques et radiologiques des chemodectomes de la tête et du cou. Il présente ses constatations sur 30 cas. Il insiste sur l'importance des différentes méthodes radiologiques dans le diagnostic de ces lésions.

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## RESEAU ADMIRABLE CAROTIDIEN HUMAIN

### Problemes etiology et nosologiques

par

L PICARD J M ANDRE M RENARD, J MONTAUT et P TRIDON

Au cours de ces dernieres annees de tres nombreuses observations concernant des aspects angiographiques «locateurs» de malformations angiomateuses non arterioveineuses ont ete publiees dans la litterature mondiale. De telles observations recouvrent en fait des affections fort disparates allant des malformations congenitales ressemblant aux «reseaux admirables» de l'animal tout a fait exceptionnels chez l'homme a une affection acquise encore mal definie particulierement frequente au Japon au cours de laquelle des stenoses multiples iteratives des arteres de moyen calibre aboutissent a la formation d'un reseau capillaire anormal jamais rencontre dans les stenoses atheromateuses en passant par quelques malformations arterio-arterielles proximales du systeme carotidien dont l'origine congenitale ou acquise reste discutee. Ces reseau capillaires cerebraux anormaux constituent un cadre nosologique nouveau que nous nous proposons de preciser a la lumiere des cas actuellement publies et a partir de 14 observations personnelles.

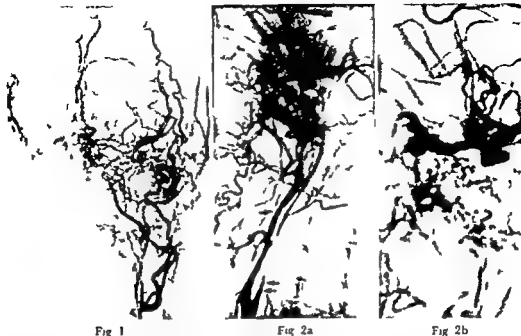


Fig 1

Fig 2a

Fig 2b

**Fig 1** Cas 1 Angiographie carotidienne gauche de face (soustraction) Ce cliché montre l'aspect hypoplasique de la carotide interne qui contraste avec la dilatation des branches de la carotide externe. On distingue surtout le réseau capillaire malformatif qui s'étend entre la terminaison des artères carotides interne et externe et le départ des artères cérébrales antérieures sylviennes après avoir entouré la région sellaire.

**Fig 2** Cas 2 a) Angiographie carotidienne gauche (soustraction) Ce cliché de profil montre la dilatation de la carotide externe (—>) et de ses branches alors que la carotide interne est très grêle (≡≡). Ces deux dernières artères donnent naissance à un réseau capillaire malformatif d'allure angiomateuse d'où s'échappent l'artère ophtalmique et la partie terminale du siphon carotidien ou sont appendus de nombreux anévrismes. Cette malformation donne ensuite naissance à la cérébrale antérieure et à la sylvienne. Il n'y a aucune image de shunt artério-veineux anormal. Le cliché agrandi (b) objective mieux le réseau capillaire malformatif et les nombreux anévrismes de l'artère ophtalmique et du siphon carotidien.

### *Les réseaux capillaires cérébraux malformatifs ou dysembryogénétiques — « réseaux admirables » vrais*

**Cas 1** Enfant origine française 6 ans acrocephalosyndactylie d'Apert, débilité mentale profonde, hypertension artérielle par sténose des deux artères rénales. Troubles anoxiques néonataux. Angiographie (Fig 1) carotides externes volumineuses et carotides internes d'aspect « hypoplasique » donnant naissance à un réseau capillaire péricallaire d'où s'échappent les deux artères péricallaires et les deux groupes sylviens. Retard circulatoire très important. Retour veineux dans un territoire profond normal. Petite malformation vasculaire occipitale injectant aux dépens des rameaux de la mésentérique moyenne. EGF légère dilatation globale des cavités ventriculaires. Caryotype normal.

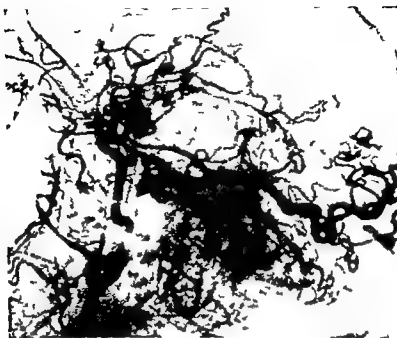


Fig 3 Cas 3 Angiographie carotidienne gauche de profil (soustraction). Cet examen montre un réseau angiomateux développé « à partir du tronc de l'artère sylvienne. Ce réseau donne ensuite naissance aux différentes artères du groupe sylvien. La choroidienne antérieure est dilatée de même que la cérébrale postérieure qui participe à la revascularisation du territoire sylvien par l'intermédiaire des artères choroidiennes postérieures et des anastomoses leptomeningées.

Cas 2 Femme : origine française 37 ans antécédents de tuberculose pulmonaire de lithiase biliaire d'avortements spontanés Céphalées depuis l'âge de 12 ans Crises convulsives généralisées à partir de 16 ans Strabisme ancien paralytique interne gauche Fond d'œil normal à droite choroidose myopique à gauche Angiographie (Fig 2) carotides externes dilatées et carotides internes « d'aspect hypoplasique » donnant naissance à un réseau capillaire d'allure angiomateuse d'où s'échappent l'artère ophtalmique la partie terminale du siphon carotidien (ou sont appendus, de nombreux anévrysmes) puis les artères pericalluses et sylviennes Important retard circulatoire Retour veineux tardif mais normal.

Ces réseaux cérébraux anormaux malformatifs réalisent un système compact de vaisseaux très fins tortueux, librement anastomosés situés à proximité du cercle de Willis dans la région parasellaire et sont constitués aux dépens des artères carotides externes et internes la carotide interne « un aspect hypoplasique alors que la carotide externe est au contraire extrêmement développée et



Fig. 4. Cas 4. a) Angiographie carotidienne gauche de profil (soustraction). Ce cliché montre un rétrécissement très serré de la partie terminale du siphon carotidien qui donne en suite naissance à un réseau capillaire anormal d'allure angiomateuse d'où se chiappent la cérébrale antérieure et les différentes branches du groupe sylvien qui paraissent élargies et spasmées. Les différentes branches de la carotide externe sont dilatées. b) Angiographie carotidienne gauche de face (soustraction). La carotide cervicale a un calibre réduit. Le siphon carotidien est rétréci en l'éc de flûte pour donner naissance au réseau capillaire anormal profond. Les artères choroïdiennes ont aussi le même calibre que l'artère ophtalmique qui est normale.

dilatée ainsi que la plupart de ses branches. Le réseau capillaire anormal donne ensuite naissance aux artères pericalluses et sylviennes. Ces véritables « réseaux admirables » (rete mirabile) fréquents chez certains mammifères (DANIEL et coll 1953) semblent exceptionnels chez l'homme à notre connaissance. Jusqu'à présent, trois cas ont été rapportés. L'un par MINAGI & NEWTON (1966) et les deux autres (ARNOLD et coll 1967).

Cliniquement, la première observation (MINAGI & NEWTON) se présentait comme une sclérose en plaques, la seconde comme une débilité avec hémiparésie droite, la troisième comme une comitialité tardive avec céphalées violentes. Dans nos deux cas, il existait un syndrome polymalformatif (acrocephalo-syndactylie d'Apert, anévrysmes cérébraux multiples).

L'origine dysembryogénétique appuyée par le contexte malformatif n'est en fait qu'une supposition puisque un tel aspect n'est jamais rencontré au cours du développement embryologique normal. À propos de ces observations, LAZORTHES

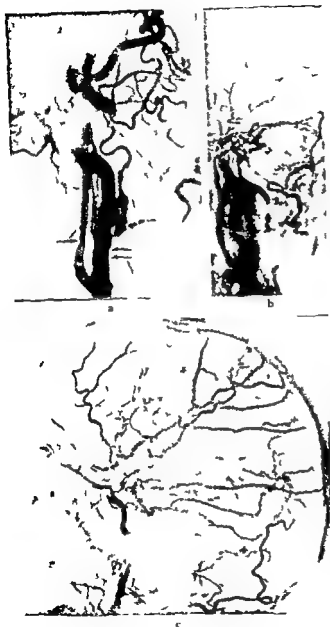


Fig 5 Cas 5 a) Angiographie carotidienne gauche centrée sur la région cervicale (soustraction) Dilatation considérable de la carotide externe ( $\times$ ) contrastant avec l'aspect hypoplasique de la carotide interne ( $\rightarrow$ ) b) Angiographie carotidienne gauche de face (soustraction) Sténose serrée de la partie terminale du siphon carotidien Absence d'injection de l'artère sylvienne et injection très faible de l'artère pericallosale gauche qui est très grêle c) Angiographie carotidienne gauche de profil (soustraction) Ce cliché objective mieux la sténose de la partie terminale du siphon d'où s'échappe un réseau capillaire anormal qui donne naissance à une artère pericallosale très grêle L'artère ophtalmique située en aval de la sténose est anormalement dilatée Les différentes branches de la carotide externe sont dilatées et donnent naissance à des pelotons vasculaires d'allure angiomateuse constituant des anastomoses avec les branches cortico-méningées de la carotide interne

& GOUAZE ont envisagé la possibilité d'un « dérapage lors du développement vers une disposition rencontrée chez certains mammifères au dessous des primates »



### *Les réseaux capillaires cérébraux anormaux et ouquant un angiome artériel*

*Cas 3* Homme origine française 43 ans sans antécédent pathologique. Hémorragie méningée cataclysmique avec hémipléxie droite. Angiographie carotidienne gauche (Fig. 3) : réseau vasculaire anormal développé à partir du tronc de l'artère sylvienne d'où s'échappent les différentes branches artérielles. Dilatation de la choréodermie antérieure et de la cérébrale postérieure. Impression anastomose leptoméninges. Carotide est une normale. Évolution rapidement fatale. Pas de vérification anatomique.

Les progrès de l'angiographie ont fait s'amenuiser et puis peu à peu disparaître le cadre des angiomes artério-artériels purs antérieurement appelés « angiomes racemeux ». L'observation que nous venons de rapporter pose à nouveau le problème. Elle n'a en effet aucun point commun avec les réseaux admirables précédemment décrits. Elle paraît différente des réseaux capillaires développés à la suite de sténoses multiples que nous allons envisager étant données l'absence de sténose et le caractère très localisé des lésions artérielles.

### *Les réseaux capillaires cérébraux anormaux — sténoses progressives multiples*

*Cas 4* Femme originaire polonaise 37 ans sans antécédent pathologique. Hémorragie méningée sous-arachnoïdienne. Signes d'infarctus pyramidal gauche. Angiographie carotidienne bilatérale (Fig. 4) : rétrécissement en « bec de flûte » des siphons carotidiens qui donnent naissance à un réseau vasculaire anormal d'allure angiomateuse d'où s'échappent les artères pericallosales et sylviennes. Le système artériel vertébro-basilaire est normal et les carotides externes sont dilatées. La carotide interne présente un aspect grêle dans son trajet cervical. Évolution favorable. Aucun séquell un an plus tard.

*Cas 5* Homme originaire française 47 ans. Hémorragie méningée avec inondation ventriculaire. Coma profond. Hémipléxie gauche. Signe de Babinski bilatéral. Angiographie carotidienne droite (Figs 5-6) montrant une sténose des artères du groupe sylvien avec un réactif déplaçant les structures médianes. Angiographie carotidienne gauche montrant un carotide interne d'aspect hypoplasique par rapport à un carotide externe dilaté. Sténose de la partie terminale du siphon carotidien. Dilatation des artères méningées avec peloton vasculaire d'allure angiomateuse correspondant vraisemblablement à des anastomoses entre la carotide externe et les branches cortico-méningées de la carotide interne.

Les réseaux capillaires cérébraux anormaux acquis se caractérisent par une sténose bilatérale plus ou moins stricte de la portion terminale des carotides internes, pouvant s'étendre au segment initial des pericallosales des sylviennes des communicantes postérieures et même intéresser la cérébrale postérieure, voire le tronc basilaire (50 % des cas pour Suzuki). La revascularisation des artères cérébrales antérieures et moyennes est assurée respectivement par un réseau vasculaire fin dense et complexe (« moyamoya », « fibrilla like vessels ») naissant des carotides internes et des communicantes postérieures par les branches perforantes — considérablement dilatées —, par les anastomoses leptoméningées

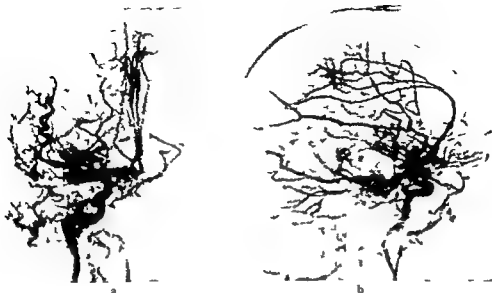


Fig. 11 Cas 5 a) Angiographie carotidienne droite de face (soustraction) Ce cliché montre lui aussi un réseau capillaire anormal profond se projetant en regard du pont de d part des artères lentulo-striées Sténose partielle des artères du groupe sylvien Œdème réactionnel déplaçant légèrement la pericalléuse vers la gauche b) Angiographie carotidienne droite de profil (soustraction) Sténose partielle de certaines artères du groupe sylvien avec œdème réactionnel sans dilatation anormale des branches de la carotide externe

si la cérébrale postérieure reste perméable « ramus spleni » choroidienne postérieure) et peut être ce qui ne nous semble pas formellement démontrer par des anastomoses transdurales unissant les territoires des carotides externe et interne, qualifiées de « rete mirabile » par de nombreux auteurs terme de toutes façons impropre (LIE 1968)

Décrite pour la première fois par TAKEUCHI en 1961 l'affection frappe surtout les jeunes japonais KUDO en recensait 146 cas en 1966 NISHIMOTO 117 cas en 1961 Vingt huit observations seulement concernent des sujets non japonais (LEEDS 1965 POOL 1967 TAVERAS 1969 aux U.S.A. SIMON et coll 1968 BUSCH 1969 et nos deux observations N° 4 et N° 5 en France)

A la suite de l'observation de KUDO confirmée depuis par de nombreuses constatations identiques il est possible d'affirmer le caractère acquis de ces anomalies vasculaires souvent évolutives susceptibles de tabilisation voire même d'amélioration ainsi SUZUKI peut-être à même de distinguer six stades successifs

Le tableau clinique protéiforme est peu évocateur accidents hémorragiques (hématomes sous-duraux dits « spontanés hémorragies méningées » accidents



Fig 7 Cas 6 a) Angiographie carotidienne gauche de profil (soustraction) Stenose à l'extrémité distale de l'artère pericalluse ainsi que des différentes branches du groupe sylvien à partir desquelles se développe un réseau capillaire anormal diffus Dilatation importante des branches choroidiennes b) Angiographie carotidienne gauche de face (soustraction) Ce cliché objective mieux la stenose de l'artère sylvienne qui donne ensuite naissance au réseau capillaire anormal Il existe un léger déplacement des branches terminales de la pericalluse probablement en rapport avec un œdème réactionnel c) Angiographie vertébrale gauche de profil (soustraction) Le processus sténosant atteint les artères cérébrales postérieures ainsi que les artères cérébelleuses antéro-supérieures et postéro-inférieures

ischémiques récidivants multifocaux. À côté de ces formes on connaît quelques cas unilatéraux (NISHIMOTO, TAVERAS) ne possédant aucun caractère propre et qui pourraient correspondre aux formes de début.

L'observation suivante est digne du groupe précédent par sa topographie lésionnelle.

**Cas 6** Femme, origine française, 32 ans. Antécédents marqués par deux avortements spontanés. Hémiplegie gauche au décours d'un curetage utérin. Un an plus tard, syndrome confusionnel aigu, LCR normal. Scintigraphie cérébrale : hyperfixation superficielle diffuse, stable dans le temps. Bilan biologique sans anomalie. Angiographie carotidienne droite (Figs 7-8) de face : thrombose de l'artère sylvienne, réseau vasculaire anormal à partir du siphon. Réseau identique se développe aux dépens des artères lenticulo-striées. Retour veineux tardif de profil : stenose pericalluse et callosa marginale à partir desquelles se développe un réseau capillaire anormal s'anastomosant avec le réseau sylvien et s'étendant sur toute la convexité. À gauche : carotide interne grêle, stenose sylvienne à 4 cm de son origine, artères en candelabre très grêles donnant naissance à un réseau capillaire anormal très riche.

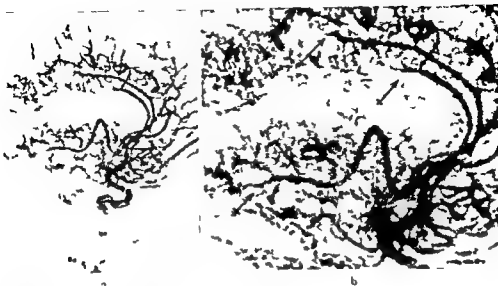


Fig 8 Cas II a) Angiographie carotidienne droite de profil (soustraction) Stenose des artères pericallosale et callosomarginale associée à une sténose sylvienne et formation d'un réseau capillaire anormal anastomosant les deux territoires b) Le cliché agrandi montre le détail des sténoses de l'artère callosomarginale (→) de l'artère pericallosale (↔) et de l'artère sylvienne (↗)

ment anastomose avec les branches de la pericallosale sténosée en « bec de flûte » à la jonction fronto-pariétale. Choroi-dienne postérieure extraordinairement développée retard circulatoire considérable. Drainage veineux normal. Angiographies vertébrales : sténose des cérébrales postérieures après le départ des choroïdiennes, dilatation des vaisseaux thalamiques qui participent à la formation du réseau capillaire anormal profond.

Les carotides internes et en particulier, les siphons sont normaux. Par contre les artères de petit et de moyen calibre sont oblitérées en « bec de flûte » dans leur portion initiale. Le réseau vasculaire d'aspect identique au « moyamoya » est lui aussi plus distal. Dans quelques cas antérieurement rapportés, les artères cérébrales moyennes et pericallosales semblent « gommées » sur un très court segment de leur trajet, jamais cependant le réseau n'atteint l'importance de celui rencontré ici. Quoi qu'il en soit par son histoire clinique et par son aspect angiographique, cette observation semble devoir s'inscrire dans le même cadre nosologique.

La pathogénie de ces réseaux capillaires cérébraux anormaux acquis est inconnue. Chez nos malades, de nombreuses étiologies ont pu être éliminées : athérome, surcharge métabolique (hyperuricémie, diabète), élastopathies systématisées (maladie de Marfan, élastorhexie), processus inflammatoire ou infec-

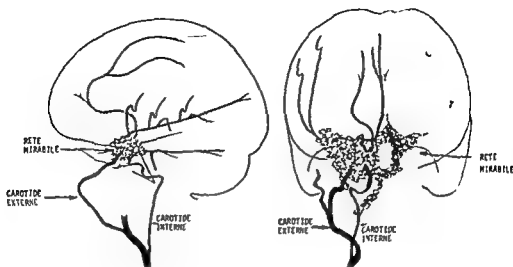


Fig. 9 Schéma du « rete mirabile » humain

tieux coagulopathie d'origine. Des cinq études anatomo-pathologiques pratiquées seule celle de KAWAKITA de VUJA et coll. et surtout celle de MAKI ont interprétables. Ce dernier note au niveau des carotides internes une réduction de la lumière artérielle un épaississement de toutes les tuniques infiltrées par des cellules lymphomonocytaires. Ces anomalies ont été retrouvées au niveau de tous les vaisseaux cérébraux à la différence cependant que la lumière est au contraire augmentée et la lamina elastica interne fragmentée de place en place ce qui pourrait expliquer les anévrysmes quelquefois notés. SUZUKI fait remarquer que l'on trouve très souvent chez ces patients des épisodes infectieux.

A la lumière de ces faits on peut évoquer à l'origine de ces sténoses progressives multiples soit un processus artériopathique secondaire à une agression vasculaire, peut-être non spécifique, soit un processus inflammatoire intéressant les vaisseaux de moyen et ou de petit calibre qui pourrait se rapprocher de la maladie de TAKAYASU touchant électivement les gros troncs artériels. Le fait que ces deux affections possèdent le même géotropisme ne saurait cependant suffire pour supposer un agent étiologique sinon commun du moins voisin. On peut de plus se demander pourquoi de tels « réseaux de suppléance » ne se produisent pas à la suite d'obstructions athéromateuses. TAVRAS pense qu'une explication réside peut-être dans le fait qu'il existe souvent une sténose de toutes les branches du polygone de Willis, ce dernier ne peut alors plus jouer son rôle de « régulateur » et les branches perforantes profondes se dilatent pour former ces réseaux capillaires anormaux.

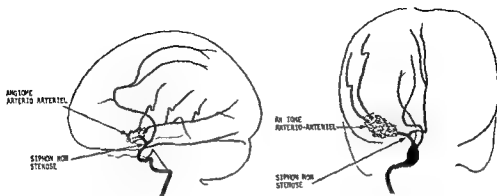


Fig 10 Angiome arterio-arteriel

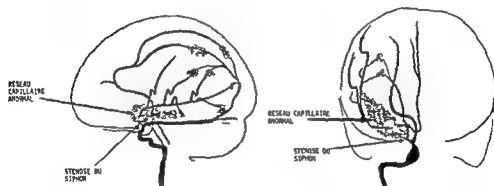


Fig 11 Schémas des réseaux vasculaires anormaux secondaires aux sténoses multiples progressives des artères intracrâniennes

### Conclusions

Ces 6 observations nous permettent d'essayer d'éclaircir les difficiles problèmes nosologiques posés par de tels aspects angiographiques. Nous pensons qu'elles représentent trois affections fort différentes de véritables « rete mirabile » (cas 1 et 2) un angiome arterio-arteriel (cas 3) et une maladie artérielle caractérisée par des occlusions multiples et progressives des artères intracrâniennes (cas 4, 5, 6).

Le véritable « rete mirabile » qui n'existe théoriquement que chez l'animal est un réseau compact de nombreux capillaires tortueux librement anastomosés situé à proximité du cercle de Willis dans la région pétilleuse. Ce réseau dépend essentiellement des carotides externes volumineuses alors que les

## CEREBRAL ANGIOGRAPHY IN FOCAL EPILEPSY

by

B. A. RING and MARGARET M. WADDINGTON

Focal epilepsy, as opposed to the centrocephalic type, is investigated neuro-radiologically because of the probability of finding gross lesions such as tumors or vascular malformations. However a small number present no definite abnormalities and although occlusive cerebrovascular disease is assumed to be responsible in many older patients (SCHMIDT & WILDER 1968), the actual occlusions are rarely seen and there are additional cases in whom cerebrovascular disease seems clinically unlikely.

Angiographic changes in patients with motor manifestations of focal epilepsy were investigated by WADDINGTON (1970) and an investigation is being continued on all focal seizures. The present report is based on 34 patients and is divided into those with motor seizures and those with focal epilepsy in general although the only differences between the two appear to be in the location of the lesion.

The angiographic changes in patients with focal motor seizures are confined to the motor or premotor areas and a detailed knowledge of local arterial anatomy is essential in recognizing alterations. The motor area on the convexity of the brain is supplied by the anterior component of either one larger or two smaller arteries running in the central sulcus. The motor strip medially is supplied by the arteries

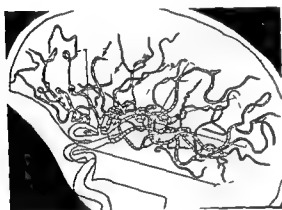
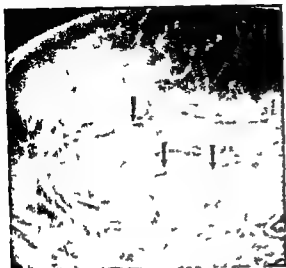
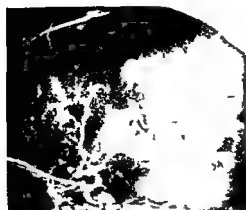


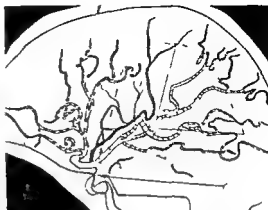
Fig 1 a) Photographic enlargement of anterior portion of carotid angiogram on a 77 year-old man with focal motor seizures and facial weakness following an acute stroke like episode. Arrows point to occlusions of three small branches in the premotor and motor areas b) Drawing of the entire angiogram, with only the middle cerebral artery branches shown. The solid vessels are the blood supply to the motor and sensory areas. The stippled area represents the site of both a positive brain scan and the electroencephalographic focus.

to the paracentral lobule details of these anatomical arrangements have been published previously (RING 1969 RING & WADDINGTON 1967 & 1967 b). These patients can be divided into two types with good correlation between clinical and angiographic findings. The largest group (16 patients) is composed of either older patients or of children whose seizures followed conditions predisposing to arterial thrombosis such as a severe febrile illness. These patients all had neurologic abnormalities: some had positive brain scans and cerebral angiography disclosed occlusion of tiny branches of the arteries in the motor or premotor areas.





a



b

Fig. 2 a) Angiogram of a 10-year old boy with focal motor seizures and no neurologic findings or an history suggesting a cerebrovascular accident. There is a very large central sulcus artery but the branches are directed upward and posteriorly with a relative avascular area anteriorly. b) The central sulcus arteries are in black, and the large component is seen to supply the posterior portion (the sensory strip) with very tiny branches to the motor area.



a



b



c

Fig. 3 a) Angiogram of a 9-year old boy with seizures following an acute febrile illness three years previously and a focally abnormal electroencephalogram. A neurologic deficit, consisting of speech disorder persisted. There is a gross occlusion of the posterior temporal artery. b) The point of occlusion of the posterior temporal artery is obvious and a small component of the central sulcus artery (solid black) is very small and filled slowly (dotted lines). The stippled area represents the abnormal electroencephalograph focus. Brain scan was negative. c) Encephalography. Despite the occlusion of a fairly large artery there is only minimal asymmetry of the ventricles without localized atrophy. (Courtesy of John McAfee, Department of Radiology, Upstate Medical Center, Syracuse, NY.)

Of this group 9 patients had occlusions of tiny branches of the central sulcus artery, in 3 there were occlusions of small branches of both central sulcus and operculofrontal arteries in 2 only the operculofrontal arteries, supplying the premotor area were involved and in 2 the occlusions involved a branch supplying the anterior portion of the paracentral lobule. It should be emphasized that all these occlusions were of very small branches. Many of these occlusions in older individuals appeared to be embolic. These patients can be described as having mini strokes that differ from ordinary occlusive cerebrovascular disease only in the size of the vessel involved. An example of this condition is shown in Fig. 1.

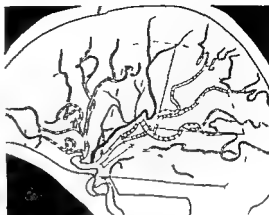
The second smaller group of 7 patients did not appear to have had vascular occlusions and for want of a better name are described as having a perfusion deficit. These patients were older children or adults and clinically had no neurologic abnormalities. The findings in these patients were limited to an unusually small arterial branch to the motor area. In 6 patients, the anterior component of the arteries in the central sulcus were involved. In one with leg seizures there was an unusually small artery supplying the paracentral lobule. These changes are characteristic but since we have seen two patients with similar small arteries to the motor area without seizures they cannot be considered specific. An example of this type is shown in Fig. 2.

From current material 11 cases with focal epilepsy and no gross angiographic abnormalities have been recorded. The majority are similar to those in the focal motor category in that they are primarily older patients and have small intracranial occlusions. Unlike the focal motor group, the occlusions are found anywhere in the brain except the motor areas and the occlusions, although small involve larger branches than those with focal motor seizures but unlike those with focal motor seizures neurologic deficit was rare. None of these patients had positive brain scans. Fig. 3 is an example of this type.

Three patients had no actual occlusions but did have unusual avascular areas perhaps comparable to the small arteries to the motor areas in the focal motor group. One was aged 6 the others aged 21 and 23, and none had neurologic abnormalities or a history suggestive of stroke or any positive findings other than the abnormal EEG. One patient was examined by cerebral angiography three years apart with no demonstrable change in the avascular area. In one 29 year old female the avascularity was obvious consisting of total absence of the angular artery such as is ordinarily associated with a definite stroke syndrome and persistent neurologic deficit (Fig. 4). We have seen one similar case mentioned in a report on intracranial arterial occlusion in 1963 (RINE). This patient had a convulsive disorder and complete absence of the posterior parietal artery. There was no clinical evidence of stroke and the case was dismissed as a probable false positive.



a

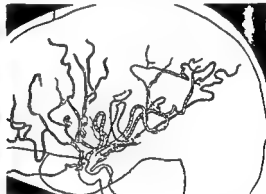


b

Fig 2 a) Angiogram of a 10 year old boy with focal motor seizures and no neurologic findings or any history suggesting a cerebrovascular accident. There is a very large central sulcus artery but the branches are directed upward and posteriorly with a relative avascular area anteriorly. b) The central sulcus arteries are in black and the large component is seen to supply the posterior portion (the sensory strip) with very tiny branches to the motor area.



a



b

Fig 3 a) Angiogram of a 9 year old boy with seizures following an acute febrile illness three years previously and a focally abnormal electroencephalogram. A neurologic deficit consisting of speech disorder persisted. There is a gross occlusion of the posterior temporal artery. b) The point of occlusion of the posterior temporal artery is obvious and a small component of the central sulcus artery (solid black) is very small and filled slowly (dotted lines). The stippled area represents the abnormal electroencephalographic focus. Brain scan was negative. c) Encephalography. Despite the occlusion of a fairly large artery there is only minimal asymmetry of the ventricles without localized atrophy. (Courtesy of John McAfee, Department of Radiology, Upstate Medical Center, Syracuse, NY.)



c

compromised areas. However this is speculative and we can only as a conclusion, that patients with focal seizures do have alterations in the cerebral arteries that if not specific are at least characteristic.

### SUMMARY

Focal epilepsy as opposed to the centrocephalic type is frequently associated with gross abnormalities such as tumors or vascular malformations. Angiograms of 34 cases in whom no gross abnormalities were present demonstrated occlusions of small cortical arteries in the majority with a smaller number having only small or absent arterial branches at the site of the focus. Lesions involving the motor or premotor area present as focal motor seizures.

### ZUSAMMENFASSUNG

Die herdbedingte Epilepsie ist im Gegensatz zum centrocephalen Typ häufig mit groben Veränderungen wie Tumoren oder vaskulären Missbildungen verbunden. Angiogramme bei 34 Fällen, bei denen keine groben Veränderungen vorhanden waren, zeigten Okklusionen der kleinen corticalen Arterien, in der Mehrzahl mit einer geringeren Anzahl entweder kleinerer oder fehlender arterieller Äste im Bereich des Herdes verbunden. Schädigungen, bei denen die motorischen oder prämotorischen Gebiete betroffen sind, kommen in herdbedingten motorischen Anfällen zum Ausdruck.

### RÉSUMÉ

L'épilepsie focale contrairement à l'épilepsie centrencephalique est souvent associée à des lésions volumineuses telles que des tumeurs ou des malformations vasculaires. Les angiographies de 34 cas où il n'y avait pas de lésions volumineuses ont montré des occlusions de petites artères corticales dans la plupart des cas. Dans un plus petit nombre de cas les angiographies ont montré au siège du foyer épileptogène soit seulement de petites artères soit l'absence de branches artérielles. Les lésions qui touchent l'aire motrice ou l'aire prémotrice donnent lieu à des crises focales motrices.

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## TOPOGRAPHIC INVESTIGATION OF THE CORTICAL BRANCHES OF THE MIDDLE CEREBRAL ARTERY

Their identification by angiography

by

G SALAMON, J GONZALES, J FAURE and G GIUDICELLI

Anatomic investigations of the cortical branches of the middle cerebral artery have been performed by DURET MOUCHET and FOIX & LEVY and others and in more recent times by GABRIELLE et coll DUBOUX et coll LAZORTHES and JAIN. Angiography of these vessels has however presented certain difficulties. This is reflected in the fact that the schematic representations of lateral carotid angiograms in many textbooks of neuroradiology mention neither the central nor the parieto occipital (angular) arteries. The procedure proposed by RING & WADDINGTON whereby the areas of the different cortical branches of the middle cerebral may be outlined in angiograms, represents an interesting new approach, interesting because of the help it may afford in the diagnosis of partial thrombosis of this artery or in the detection of minor displacements of its cortical branches.

The purpose of this communication is to describe a system probably more accurate than any hitherto proposed for identifying these cortical branches.

*Anatomic technique* Twenty brains in which the middle cerebral artery had been injected with an opaque substance were dissected. The procedure was simple and consisted in the introduction under image intensifier control of a small amount of red lead mixed with gelatin. After fixation for a month, the hemisphere was then photographed and, after metal wires had been positioned to

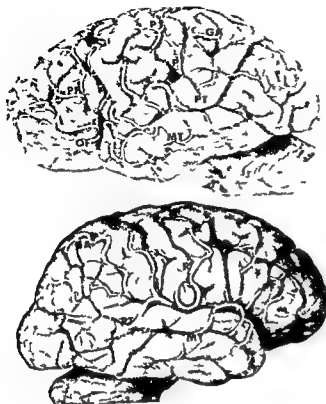


Fig 1 Dissection of middle cerebral artery OF — orbitofrontal artery PF — prefrontal artery PR — precentral artery R — central artery P — parietal arteries GA — parieto-occipital (angular) artery PT — posterior temporal artery MT — middle temporal artery AT — anterior temporal artery

mark the anterior and posterior commissures was roentgenographed (Fig 2) A horizontal wire through the upper border of the anterior commissure and the lower border of the posterior commissure marked the inter commissural base line two vertical wires at right angles to the horizontal wire, passed through the centres of the anterior and posterior commissures respectively This procedure which TALAIRACH & SZIKLA introduced for stereotactic identification, enables tracing to be made from photographs or roentgenograms of each cortical branch of the middle cerebral artery (Figs 5 to 8) An examination of the tracings had indicated that the course of each of these vessels is relatively constant in relation to the commissural guide lines

*Angiographic application* The position of cerebral structures sulci or blood vessels may be determined by reference to specific landmarks on the skull However because of the differences in the development of the brain and skull respectively this method is fraught with inaccuracy

TALAIRACH & SZIKLA demonstrated that the anterior and posterior commissures bear a constant topographic relationship to a line joining the jugum



Fig 2 Roentgenogram of a cerebral hemisphere with middle cerebral artery injected 1 — intercommisural base line passing through the anterior and posterior commissures 2 — vertical line through anterior commissure 3 — vertical line through posterior commissure (Abbreviations as in fig 1)

sphenoidale to the lower margin of the internal occipital protuberance (Fig 3). The figures of these authors have to be corrected for conventional skull roentgenograms or cerebral angiograms at a FFD of around 80 to 100 cm to allow for a coefficient of magnification not present in their measurements (obtained from teleroentgenograms). Once the corrections are made the line joining the anterior and posterior commissures may be re drawn and the position of these structures defined. The commissures may then be used as a base line from which to estimate the position of other structures.

The identification aid illustrated in Fig 4 was developed as an application of this principle and consists of a transparent plastic disk prepared as follows. A line was drawn which when laid over a carotid angiogram passed through the jugum sphenoidale and the lower margin of the internal occipital protuberance. A point 9.3 mm behind the jugum and 26.7 mm above this line located the anterior commissure. A second horizontal line was then drawn from the same point on this jugum protuberance line parallel to the first horizontal line: the posterior commissure lay on the second horizontal line, 29 mm behind the anterior commissure. Thus three lines pass through the commissures: a horizontal line which is a guide to the commissures in the horizontal plane, and two vertical lines which indicate the anterior and posterior commissures respectively in the coronal plane. The conditions under which the cortical branches of the middle cerebral artery were analyzed in the dissected preparations were thus fully reproduced.

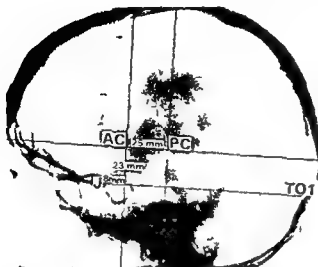


Fig 3 Method for radiologic identification of the anterior and posterior commissures (AC and PC) and the commissural reference lines (teleroentgenogram) J — jugum, IOP1 — lower margin of internal occipital protuberance (From TALAIRACH & SZIKLA plate 30)

The most frequently observed course of each cortical branch of the middle cerebral artery was plotted on a transparent plastic disk marked in the way as described. This afforded a handy instrument for rapid arterial identification and one of great accuracy, being based on a large series of anatomic dissections. Tested on 100 normal cerebral angiograms it permitted ready identification of the precentral artery, the central artery, the two parietal arteries, the parieto-occipital (angular) artery, and the posterior and middle temporal arteries in 96 instances. Difficulty was experienced in identifying the prefrontal and orbito-frontal arteries in the former because of its tripod branching and in the latter because of superimposition of the roof of the orbit.

### Comments

The method confirms the validity of a procedure whose accuracy in estimating the position of cerebral structures has already been demonstrated by TALAIRACH & SZIKLA in its application to stereotactic surgery. A positioning technique based upon the anterior and posterior commissures, the true fixed points of the brain around which embryologic development takes place, must by its very nature be more reliable than any other. The reconstruction of cerebral structures identified from their vessels, cisterns and sulci may even be possible. This is of course the essential principle of stereotactic surgery of the telencephalon.

As the method is based upon the dissection and roentgenography of anatomic preparations, the possibility of inaccuracy is reduced to a minimum, it is certainly less than in roentgen identification techniques depending upon the statistical analysis of large numbers of angiograms. It is sometimes difficult even in



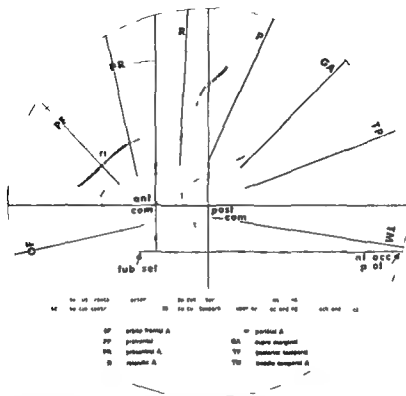


Fig 4 Plastic orientation disk for identifying the cortical branches of middle cerebral artery in carotid angiograms (Abbreviations as in fig 1)

dissections to recognize certain cerebral sulci and their corresponding vessels and consequently the improbability of angiographic analysis even if based on hundreds of films achieving comparable precision will be appreciated.

The method is quick — it gives the desired result in a few seconds — and is therefore of practical utility. It is precisely because they take so long that the complicated procedures that have from time to time been proposed for measuring a p or p a displacement of the pineal body or for determining the position of the central sulcus are little used in routine work. A means of rapid definition of the cortical branches of the middle cerebral artery and a knowledge of their respective topographies should be of service in the investigation of cerebral vascular accidents since fewer partial thromboses will escape detection. It should also be useful in the diagnosis of intracranial expanding processes as the location of limited lesions will be facilitated.

The transparent plastic disk described in this paper may be obtained from the authors. Requests should be addressed to Prof G Salamon, Department of Neuroradiology, Hôpital de la Timone, Boulevard Jean Moulin, 13 Marseille, France.

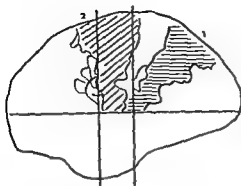


Fig 5 Course of (1) parieto-occipital (angular) branch and (2) central branch of middle cerebral artery

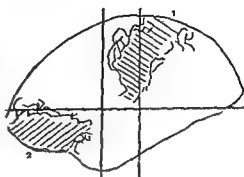


Fig 6 Course of (1) parietal branch and (2) orbitofrontal branch of middle cerebral artery

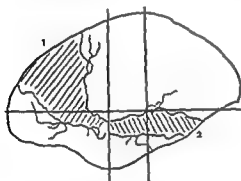


Fig 7 Course of (1) prefrontal branch and (2) middle temporal branch of middle cerebral artery

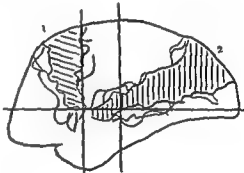


Fig 8 Course of (1) precentral branch and (2) posterior temporal branch of middle cerebral artery

## SUMMARY

The middle cerebral arteries of twenty brains were injected selectively, dissected out and the brains roentgenographed. The course of each cortical branch of the middle cerebral artery was plotted from the data on a special orientation disk. Angiographic analysis based upon this method the accuracy of which was confirmed by 100 normal angiograms is proposed.

## ZUSAMMENFASSUNG

Die mittleren Cerebralarterien von 20 Gehirnen wurden selektiv injiziert, disseziert und roentgenographiert. Der Verlauf aller corticalen Äste der mittleren Cerebralarterie wurde auf einer speziellen Orientierungstafel dargestellt. Eine angiographische Analyse auf Basis dieser Methode deren Genauigkeit bei 100 normalen Angiogrammen bestätigt wurde wird vorgeschlagen.

## RÉSUMÉ

Les auteurs ont injecté sélectivement les artères cérébrales moyennes de vingt cerveaux, les ont dissequés et ont radiographié les cerveaux. Le trajet de chaque branche corticale de l'artère cérébrale moyenne a été représenté d'après les résultats de ces examens sur un disque d'orientation spécial. Les auteurs proposent une analyse angiographique basée sur cette méthode dont la précision a été confirmée sur 100 angiographies normales.

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## ROENTGENMICROGRAPHY OF THE CEREBRAL ARTERIES IN INTRACRANIAL EXPANDING LESIONS

by

G SALAMON and C RAYBAUD

The results of the post mortem roentgenmicrographic examination of the brains of 15 patients with malignant gliomas of the cerebral hemispheres have been compared with the findings at angiography performed during their lives

Specimens for roentgenmicrography of the cerebral arteries were prepared by the slow injection over four hours of Micropaque suspension 10 % through plastic catheters placed in the carotid and basilar arteries of the brains removed immediately after death. The brains were fixed in formalin for two months following which 1 cm coronal or sagittal sections were cut. All the sections were based on the intercommissural lines so as to facilitate comparison between those from different subjects and pathologic and normal brains of the reference series. A more detailed analysis of the arteriolar network required thinner sections (800 to 1 500  $\mu$ ). Roentgenmicrography was performed with a fine focus (0.3 mm) water cooled tube (which allows relatively long exposure times) and Kodak M emulsion or Heliogul film. All the tumours were examined histologically.

*Roentgenmicrographic findings* Attention was directed to the vascularization of the cortex and white matter of the hemispheres in general and of the peritumoural zone in particular. The intratumoural angio-architecture which has

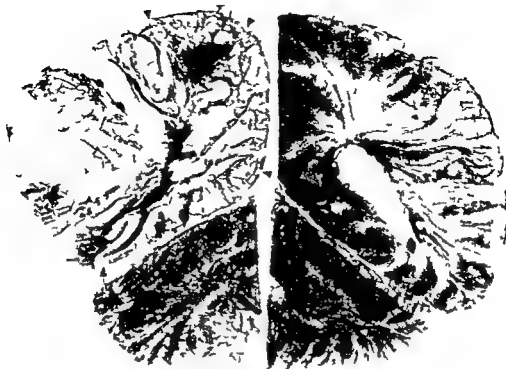


Fig. 1. Expanding lesion in parietal lobe. Normal cortical arteries compressed throughout the entire hemisphere. Few vessels present in the cortex and white matter (Compare with the opposite side).

already been the subject of extensive neuropathologic investigation was ignored.

A constant finding in tumours situated near the surface of a hemisphere and accompanied by increased intracranial pressure was a striking reduction in the arterioles of the cortex and white matter in the vicinity of the lesion; this was sometimes apparent as well in more extensive areas of the cranial vault or the falx cerebri. Diminution of the arteriolar network was greatest in the temporal cortex above the floor of the middle cranial fossa in temporal expanding lesions. Accumulations of contrast medium around a tumour corresponded to areas of softening evident in histologic sections; the medium not infrequently flowed from these areas of softening into the veins of the white matter. This venous filling was a pathologic phenomenon as control experiments in animal and human brains indicated that under normal conditions this special injection technique does not force the contrast medium into the cerebral veins.

The cortical arterial network was little affected in deep-seated tumours; in the basal ganglia, for example, even when accompanied by raised intracranial

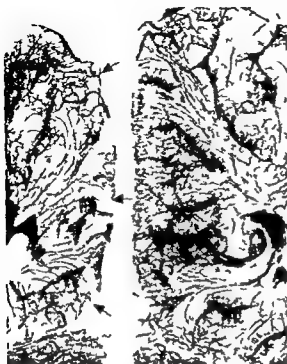


Fig 2 Expanding lesion in a cerebral hemisphere. The cortical vessels on the side of the tumour especially on the medial surface of the occipital lobe opposite the falx cerebri have disappeared.

pressure. On the other hand patches of the contrast medium in the necrosed areas and abnormal filling of veins of the white matter, as in the more superficial lesions were evident.

*Correlation with cerebral angiography.* Comparison of the angiographic and roentgenographic findings pointed to a correlation between slowing of arterial flow and severity of changes in the cortical arterioles. The longest angiographic times occurred in the brains that subsequently displayed the most marked compression of the cortical arterial network. Apparently in confirmation of the conclusions, carotid angiography in patients with basal ganglia tumours seldom however revealed reduced circulation rates even in the presence of greatly increased intracranial pressure. It seems that the low flow rates in intracranial expanding lesions are not a direct product of raised intracranial pressure but result rather from mechanical compression of the cortical arterial network of the affected hemisphere. The two mechanisms are of course related. There is also evidence to suggest that an early arteriovenous fistula often reflects a circulatory anomaly situated, not in the tumour itself, but in the softened areas around it. Identical appearances are encountered in cerebral softening of vas-



Fig 3 Venous filling in a necrotic area.

cular origin. Although this observation obviously does not apply to all arterio-venous fistulas in malignant gliomas, it does explain why disparity between the angiographic appearances and the size of the tumour as disclosed at operation sometimes exists.

### SUMMARY

The results of a roentgenomicrographic investigation of the cerebral arteries in a series of superficial and deep-seated malignant gliomas of the cerebral hemispheres are described. The appearances are compared with those observed in angiographies during the patients' lives.

### ZUSAMMENFASSUNG

Die Ergebnisse einer röntgenmikrographischen Untersuchung der cerebralen Arterien wurden bei einer Serie von oberflächlichen und tiefliegenden malignen Gliomen der cerebralen Hemisphären beschrieben. Ihr Erscheinungsbild wird mit dem, das bei angiographischen Untersuchungen dieser Patienten während des Lebens beobachtet wurde, verglichen.



Fig 4 Deep tumour Unlike in superficial tumours the peripheral vascular vessels are relatively intact

## RÉSUMÉ

Description des résultats d'une étude micro radiographique des artères cérébrales dans une série de gliomes malins superficiels et profonds des hémisphères cérébraux. Les aspects micro radiographiques sont comparés avec ceux qui ont été observés dans l'angiographie du vivant du malade.

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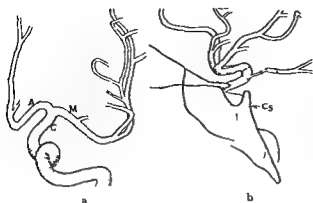


Fig 1 Diameters of main cerebral arteries a) Frontal view A = anterior cerebral artery M = middle cerebral artery C = internal carotid artery intradural part b) Lat ral view Cs = internal carotid artery parasellar part (from GABRIELSEN & GREITZ 1970)

was present in the history. All cases already operated upon, as well as those in which neurologic symptoms were present before the trauma, were excluded with the exception of a few cases of old obvious known epilepsies. A few cases were excluded for technical reasons such as gross movement blurring unusually high magnification due to large object film distance etc. A total of 67 cases, 47 males and 20 females, remained after this elimination. Forty four cases had subdural haematomas only. The extracerebral haematoma was complicated by the presence of an intracerebral expanding lesion interpreted as haematoma in 15 cases. Four cases of epidural haematomas — the total number occurring during the same time interval — were included for comparison. The age of the patients ranged from six to eighty four years.

The diameters of the internal carotid, anterior and middle cerebral arteries were measured at a distance of 0.5 cm from the bifurcation (Fig 1 a). The parasellar part of the internal carotid artery was also measured (Fig 1 b). The measurements were made by the method of GABRIELSEN & GREITZ (1970). Control measurements were performed at the points mentioned in a series of normal cerebral angiograms from the material of the same authors. Statistical calculation disclosed no significant systematic difference between the values of GABRIELSEN & GREITZ and those obtained by the present author, whose error of measurement was  $\pm 0.15$  mm. Regional circulation times as well as the mean cerebral circulation time were determined (GREITZ 1956, 1968). Only the measurements in Fig 1 a were performed in a few cases as the examinations were incomplete. The values of the cerebral vessel diameters and the mean cerebral circulation times were plotted against the time elapsing between the trauma and the angiographic examination, as regards this time interval, the material was arbitrarily divided into four groups.

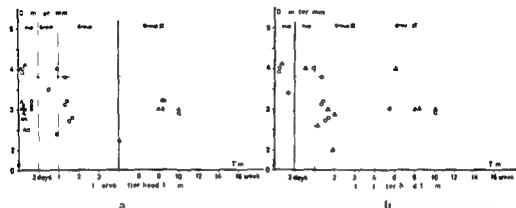


Fig 2 Temporal distribution of diameters of the middle cerebral artery. a) All cases b) After exclusion of cases with intracerebral haematoma. The horizontal lines indicate the normal mean value (heavy line) and twice the standard deviation (broken lines).  $\circ$  males and  $\Delta$  females with subdural haematoma  $\bullet$  males and  $\Delta$  females with subdural and intracerebral haematomas

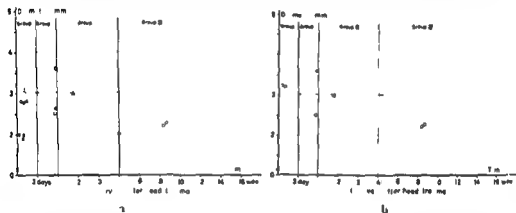


Fig 3 Temporal distribution of diameters of the anterior cerebral artery. a) All cases b) After exclusion of cases with intracerebral haematoma.  $\circ$  males and  $\Delta$  females with subdural haematoma  $\bullet$  males and  $\Delta$  females with subdural and intracerebral haematomas

The first group comprised cases examined during the first three days following the injury and was considered to represent the acute stage. The second group comprised cases in a transitional period. The third group consisted of cases examined from the beginning of the second to the end of the fourth week. The fourth group was made up of cases in the chronic stage. Most cases had only undergone angiography once although at random intervals after the cranial trauma. The temporal distribution of the cases made the division into the groups described permissible.

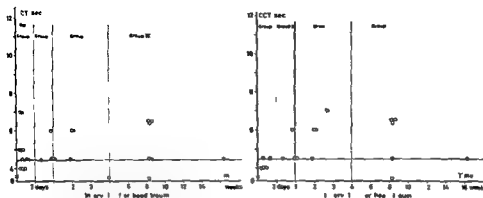


Fig 4 Temporal distribution of cerebral circulation times (CCT) The horizontal line indicates twice the standard deviation above the normal mean value (from GRITZ 1968) ○ subdural haematoma, ● subdural and intracerebral haematomas

## Results

Measurements of the diameter of the middle cerebral artery generally disclosed somewhat lower values compared with the normal (Fig 2 a). In respect of time it was difficult to discern any definite pattern when cases with and without complicating intracerebral haematomas were treated together. An intracerebral haematoma was usually associated with marked narrowing of the vessels from the first day onwards. After exclusion of cases with such complication (Fig 2 b) the values during the first three days were essentially within normal limits, from the beginning of the second week a general decrease in vessel diameters occurred. A slight tendency to return to normal limits but with a considerable scattering was recorded during the final period. The difference between the values obtained during the first three days and those between the beginning of the second and the end of the fourth week was highly significant ( $p < 0.001$ ). The diameters of the anterior cerebral artery varied in the same way with time (Fig 3 a, b). The difference between the values in groups I and III was also highly significant. Measurements of the intradural and extradural parasellar part of the internal carotid artery yielded scatter diagrams that resembled those already described. However the scatter around the mean was larger and the significance of the difference between the first and third groups less ( $p < 0.01$ ).

The results of determinations of the mean cerebral circulation times are presented in Fig 4. Cases without an intracerebral haematoma had a normal, slightly or moderately prolonged circulation time during the first three days. The values were generally higher during the third and fourth periods and

again there was a slight tendency to return to normal in the late stages with considerable scattering. Cases with an intracerebral haematoma had a marked prolongation of the mean cerebral circulation time from the first day onwards. No correlation was found between the mean circulation time and the size of the haematoma. The latter varied with time in the usual way. Old haematomas were always large up to 4 cm thick while recent haematomas varied from a few millimeters to several centimeters.

Local prolongation of the circulation time often accompanied by decreased capillary filling in the same area could be observed in 29 cases, 14 of which occurred during the first three days. Nine of these were complicated by an intracerebral haematoma. When only cases with extracerebral haematomas were considered the local circulatory retardation occurred in a third to two-fifths of the cases, a frequency that did not vary significantly in respect to time.

### Discussion

The feasibility of measuring cerebral vessel diameter in angiographic films has been demonstrated earlier and the reproducibility of the values obtained was found to be good (DE BOLLAY 1968, GABRIELSEN & GREITZ 1970, HUBER & HANDA 1967). Changes in the size of the cerebral vessels with such physiologic variables as arterial blood  $pCO_2$ ,  $pO_2$  and pH have been reported (DE BOLLAY 1968, HUBER & HANDA 1967). Only in a few instances in the present investigation was it possible to obtain data about blood gases at the time of the angiography. Furthermore the material was heterogeneous in many ways as regards such important factors as level of consciousness, mode of ventilation, state of respiration and damage to other organs. A pilot investigation however, revealed only statistically insignificant variations with time intervals between the trauma and the angiography in these respects.

The results establish that the diameter of the large extracerebral conducting vessels is significantly less in cases of extracerebral — intracranial haematomas examined one to four weeks after trauma than in those examined at one to three days. Repeat examinations which could be performed in a few cases support the assumption that this difference is due to a narrowing of the vessels (Fig. 2). This has a certain resemblance to that evident in connection with subarachnoid bleeding. Common to both conditions is the occurrence of vessel constriction and its peculiarly delayed appearance. Contrary to what is observed in subarachnoid bleeding the constriction in subdural haematomas does not seem to decrease after the second week, a fact which may be explained by the continued presence, and sometimes enlargement of the haematoma. The cerebral

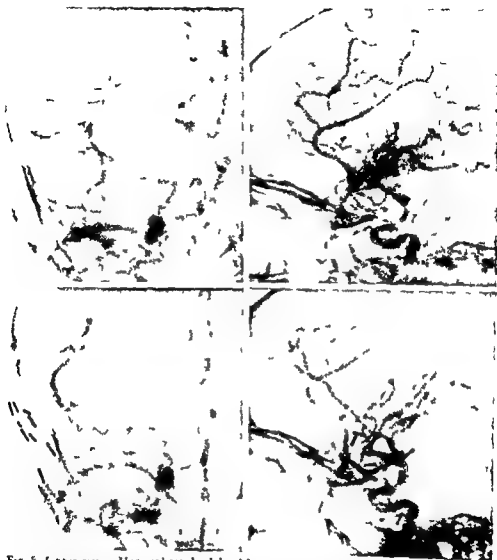


Fig 5 *Upper views* Uncomplicated subdural haematoma 7 weeks after trauma. *Lower views* The same case after operation about 2 weeks later. The general narrowing of the vessels present at the time of the first examination has disappeared.

vessels are generally narrowed from the first day following the trauma in cases complicated with intracerebral haematomas, as in cases with intracerebral haematomas only (Fig 6)

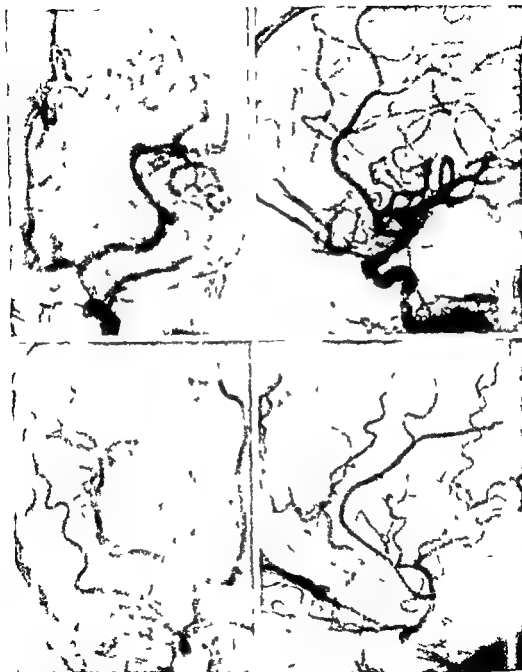


Fig. 6. *Upper views:* Acute subdural haematoma. No obvious spasm. *Lower views:* A similar case complicated with intracerebral haematoma. Marked constriction of most vessels.

The mean cerebral circulation time is prolonged in both extracerebral and intracerebral haematomas. The prolongation in its time course matches closely the pattern of the vessel size, e.g. in uncomplicated extracerebral haematomas the values of the circulation time are generally higher at one to four weeks than within three days of the trauma. The circulation time is markedly prolonged from the first day onwards in cases with extra- and intracerebral haematomas as in cases with intracerebral haematomas only.

The simultaneous decrease in cerebral vessel diameter and prolongation of circulation time suggest that a successive reduction of cerebral blood flow occurs in uncomplicated causes of subdural haematoma one to four weeks after the initial trauma. In cases complicated with an intracerebral haematoma this reduction in blood flow seems to begin as early as the first day. The generally moderate decrease in vascular diameter cannot explain but might well be the result of impairment of perfusion. The possible causes of the vessel constriction have been discussed (BRAWLEY et coll 1968, KAPP et coll 1968, POOL & POTTS 1963, WILKINS 1968) but a generally accepted explanation is still lacking. The difference observed in the behaviour of the cerebral vessels in cases with and without intracerebral haematomas suggests that more than one mechanism may be involved in bringing about the vasoconstriction. The occurrence of vessel narrowing even in cases of pure subdural haematoma (e.g. with no blood observable in the cerebrospinal fluid) clearly favours explanation that does not presuppose direct contact between blood from the haematoma and cerebral vessel adventitia. Cerebral oedema and generally increased intracranial pressure may account for or contribute substantially to the extreme prolongation of the mean cerebral circulation time with corresponding narrowing of the vessels in the acute stages of intracerebral haematomas at least in a substantial number of cases.

The early appearance of vasoconstriction and a markedly delayed cerebral circulation time in cases of head trauma may be of diagnostic importance as their occurrence suggests the presence of an intracerebral haematoma in addition to the extracerebral haematoma.

## SUMMARY

Cerebral vessel diameters and cerebral circulation time were determined angiographically in 67 cases of traumatic intracranial haematomas. The values were plotted against the time elapsing between trauma and angiography. The results suggesting a delayed appearance of diminished cerebral blood flow are described and their significance discussed. The presence or absence of intracerebral haematomas seems to influence the time course of the changes.



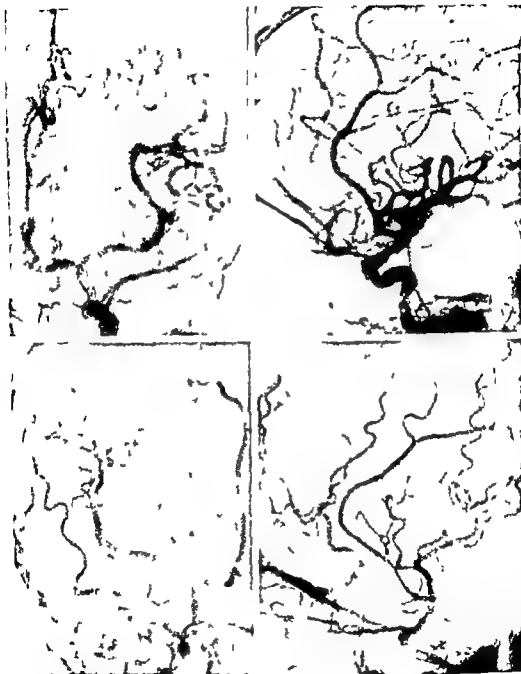


Fig 6 *Upper views* Acute subdural haematoma. No obvious spasm. *Lower views* A similar case complicated with intracerebral haematoma. Marked constriction of most vessels

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## ZUSAMMENFASSUNG

Der Diameter der Gehirngefäße und die cerebrale Zirkulationszeit wurden in 67 Fällen eines traumatischen intracranialen Hämatoms angiographisch bestimmt. Die Werte wurden gegen die Zeit, die zwischen Trauma und Angiographie lag, aufgetragen. Die Ergebnisse, die ein verzögertes Auftreten einer verminderten cerebralen Zirkulation vermuten lassen, werden beschrieben und deren Bedeutung besprochen. Das Vorhandensein oder die Abwesenheit eines intracerebralen Hämatoms scheint den Zeitablauf der Veränderungen zu beeinflussen.

## RÉSUMÉ

L'auteur a déterminé par angiographie le diamètre des vaisseaux cérébraux et le temps de circulation cérébrale dans 67 cas d'hématome intra crânien traumatique. Ces mesures ont été représentées en fonction du temps écoulé entre le traumatisme et l'angiographie. Les résultats font penser que la diminution du débit sanguin cérébral apparaît avec un certain retard. L'auteur décrit ces résultats et étudie leur signification. La présence ou l'absence d'hématomes intra cérébrales paraît influencer sur l'évolution dans le temps de ces modifications de calibre et de ces vites es circulatoires.

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Table 1

*Age and sex distribution and neurologic complications (in parentheses)*

Age	Men	Women	Total
0—9	3	1 (1)	49 (1)
10—19	31	16	47
20—29	47	32	79
30—39	53 (2)	31 (1)	84 (3)
40—49	56 (1)	38 ( )	94 (3)
50—59	45 (1)	23	68 (1)
60—69	23 (1)	10	33 (1)
70	7	1	3
Total	289 (3)	163 (4)	457 (9)

angiographies in 457 patients performed during the period 1967—1970 (Table 1). The vertebral and carotid angiographies that were not intended to demonstrate the intracranial vessels were not included. Four patients were examined on three occasions. 35 had two examinations and the remaining 418 patients each had one examination. The technique of the procedure, type of examination, size and position of the catheter, amount of contrast medium and duration of the procedure were recorded in detail at the time of examination. Neurologic investigations were performed before and after the procedures and each patient was followed for at least 48 hours for possible sequelae. Neurologic complications were defined as the worsening of old or the appearance of new neurologic signs within 24 hours following the procedure. One patient who developed fresh neurologic signs and symptoms at 30 hours was included. Arterial spasm or occlusions clearly demonstrated were also included.

The procedure was performed under light sedation and local anesthesia. A Becton Dickinson RPA 045H (outer diameter 1.65 mm) or RPA 062H (outer diameter 2.1 mm) catheter was introduced by the percutaneous femoral route with elective catheterization of the internal, external carotid and vertebral arteries. The catheters had no side holes and were of J- or S-form according to the age of the patient and the artery to be examined. The injection was made into the common carotid and subclavian arteries when elective catheterization was unsuccessful. Five to 8 ml meglumine iohalamate 60% were injected by hand or by a pressure injector (Cisal 1); the exact amount depending upon the size of the arteries. The catheter was flushed every 3 to 5 minutes with heparinized saline (500 units for 250 ml) in order to prevent clotting. Two syringes were used for flushing: one for withdrawal of blood and the other for injection of saline. The guide wire was carefully cleaned before use.

## COMPLICATIONS OF CATHETER CEREBRAL ANGIOGRAPHY

An analysis of 500 examinations

by

M. TAKAHASHI and H. KAWANAMI

Complications of carotid and vertebral angiography by the direct puncture technique, percutaneous catheterization of the common carotid arteries and retrograde brachial angiography have been evaluated and reported in detail (LINDGREN 1950, GOULD et coll 1955, FIELD et coll 1962, AMUNDSEN et coll 1963, LESTER & KLEE 1965, PERRET 1966). Only few reports of complications associated with cerebral angiography by a transfemoral catheter approach have however appeared. Furthermore, most previous publications are primarily concerned with the incidence and types of complications, more or less disregarding the major technical factors in their occurrence. The authors are of the opinion that most of these may be attributed to the angiographic technique (SCHEINBERG & ZUNKER 1963, CRONQVIST et coll 1970). It has been suggested that modern contrast media play a minor role in the sequelae (LANG 1963).

The purpose of this paper is to report the complications of catheter cerebral angiography and discuss the technical factors that may be responsible for neurologic complications. Preventive measures will also be considered.

*Material and Methods* The material comprises 500 consecutive cerebral

Table 1

*Age and sex distribution and neurologic complications (in parentheses)*

Age	Men	Women	Total
0—9	32	17 (1)	49 (1)
10—19	31	16	47
20—29	47	32	79
30—39	53 (2)	31 (1)	84 (3)
40—49	56 (1)	38 (2)	94 (3)
50—59	45 (1)	23	68 (1)
60—69	23 (1)	10	33 (1)
70	2	1	3
Total	289 (5)	168 (4)	457 (9)

angiographies in 457 patients performed during the period 1967—1970 (Table 1). Those vertebral and carotid angiographies that were not intended to demonstrate the intracranial vessels were not included. Four patients were examined on three occasions. 35 had two examinations and the remaining 418 patients each had one examination. The technique of the procedure, type of examination, size and position of the catheters, amount of contrast medium and duration of the procedure were recorded in detail at the time of examination. Neurologic investigations were performed before and after the procedures and each patient was followed for at least 48 hours for possible sequelae. Neurologic complications were defined as the worsening of old or the appearance of new neurologic signs within 24 hours following the procedure. One patient who developed fresh neurologic signs and symptoms at 30 hours was included. Arterial spasm or occlusions clearly demonstrated were also included.

The procedure was performed under light sedation and local anesthesia. A Becton Dickinson RPX 045H (outer diameter 1.65 mm) or RPX 062H (outer diameter 2.1 mm) catheter was introduced by the percutaneous femoral route with elective catheterization of the internal external carotid and vertebral arteries. The catheters had no side holes and were of J or S form according to the age of the patient and the artery to be examined. The injection was made into the common carotid and subclavian arteries when elective catheterization was unsuccessful. Five to 8 ml meglumine iohalamate 60% were injected by hand or by a pressure injector (Cisal I); the exact amount depending upon the size of the arteries. The catheter was flushed every 3 to 5 minutes with heparinized saline (500 units for 250 ml) in order to prevent clotting. Two syringes were used for flushing, one for withdrawal of blood and the other for injection of saline. The guide catheter was carefully cleaned before use.

## Results

*Complications* in catheter cerebral angiography were encountered in 13 or 2.6 per cent of examinations i.e. nine neurologic (1.8 per cent), two local (0.4 per cent) and two general complications (0.4 per cent). Arterial spasms or occlusion without neurologic symptoms or signs occurred in 20 patients or 4 per cent.

Two general and two local reactions were recorded in this series of 500 examinations. Hypotensive reaction was observed in a 44 year-old man following vertebral angiography and in a 60 year old woman who was examined by both vertebral and carotid angiography. Both patients recovered within 6 hours.

A 23 year-old woman with extensive arteritis of the aorta and brachiocephalic vessels developed thrombosis at the puncture site. No untoward sequelae were evident after thrombectomy. A 59 year old woman had massive hemorrhage at the puncture site several hours after the examination. Local complications such as local bleeding or hematomas were more frequently associated with larger sizes of catheters as well as the frequent exchange and manipulation of catheters.

There were 20 asymptomatic complications. Sixteen patients had arterial spasm of the internal carotid artery, prolonged filling of the internal carotid artery usually the carotid siphon or the segment from the tip of the catheter to the carotid siphon was evident (Fig. 1). The cerebral circulation beyond the carotid siphon was slightly slowed or was normal but no neurologic defects were apparent. This always occurred with internal carotid injection, but never with common carotid injection. The catheter tip was immediately withdrawn into the common carotid artery when these signs were observed. Similar arterial spasm of the vertebral artery without neurologic symptoms occurred in 2 patients. Two patients had occlusions of the cerebral vessels with no associated neurologic symptoms or signs. There was occlusion of the pericallosal artery in a 21 year-old man with a parietal arteriovenous malformation (Fig. 2). Postoperative angiography performed three weeks later revealed that the obstruction had cleared. An occlusion of the angular artery was noted in a 30 year-old man but was not recognized at the second injection performed 20 minutes later.

Six neurologic complications were transient and disappeared within 5 hours. Transient disorientation occurred in 3 patients, lasting for 5 hours in 1 patient and 60 minutes in 2 patients. Two of these 3 patients developed disorientation following vertebral angiography via the right subclavian catheter with three injections of 30 ml meglumine iothalamate 60%. Mild hemiparesis lasted for 5 hours in a 47 year-old woman and passing nystagmus was encountered in a 34 year old man. Tonsillar herniation developed in a 5 year-old girl following vertebral



Fig 1



Fig 2

Fig 1 Arterial spasm beyond the tip of the catheter in a 24 year-old man. Prolonged filling of the internal carotid artery from the tip to the carotid siphon.

Fig 2 Occlusion of the pericallosal artery in a 21 year-old man probably due to an embolus (arrow). No occlusion at carotid angiography 3 weeks later. An arteriovenous malformation is supplied by the contralateral pericallosal artery and the angular artery.

angiography. This patient had a fourth ventricle tumour and immediate posterior fossa craniotomy was performed. No neurologic sequelae were evident.

Two long lasting complications and one permanent complication in this series of 500 examinations were recorded. There were no fatal sequelae. The incidence is 0.6 per cent of the total examinations, or 33 per cent of all neurologic complications. The brief case histories are as follows.

### Case reports

*Case 1* This 62 year-old man admitted because of intermittent episodes of disorientation considered to be due to cerebral ischemia. Cerebral angiography was performed with the percutaneous femoral catheter technique. Right internal carotid angiography was normal except for slight atherosclerosis of the internal carotid artery. The catheter was then introduced into the left vertebral artery and two injections were made. There was occlusion of the superior cerebellar artery which involved the ambient segment of this artery (Fig 3). The distal branches were reconstituted by the collateral vessels. The patient gradually developed cerebellar signs such as ataxia, adiadochokinesia, nystagmus and dysarthria for 3 days and then improved slowly over 5 months when he was just able to return to work. Further angiography was not performed.





Fig 3 Case 1 Occlusion of a short segment of the left superior cerebellar artery (arrows) in a patient with repeated attacks of cerebral ischemia. Reconstitution of the distal superior cerebellar artery.

**Case 2** A 34 year old man admitted because of several episodes of unconsciousness. Bilateral carotid angiography with a direct puncture technique disclosed atherosclerotic plaques in both the carotid siphon and the proximal parts of the anterior and middle cerebral arteries. One of the left middle cerebral arterial branches was occluded. Left vertebral angiography was performed via a transfemoral catheter technique. Occlusion of the basilar artery was apparent after the third injection (Fig 4). The patient soon developed vertigo, dysphasia, left ptosis and left facial palsy. These neurologic findings completely cleared over 5 months.

**Case 3** A 34 year old woman admitted for evaluation of episodes of seizures clinically considered to be due to cerebrovascular disease. Bilateral carotid angiography with a direct puncture technique was normal. Left vertebral angiography was performed by the percutaneous femoral catheter technique. The patient slowly developed drowsiness, slurring of speech and slow response to verbal stimuli approximately 30 hours after the examination. Within 5 hours she developed decerebrate rigidity soon followed by quadriplegia. Further vertebral angiography three weeks later revealed occlusion of the basilar artery distal to the origin of the anterior inferior cerebellar artery (Fig 5).

The age and sex of the 457 patients are presented with complications indicated in parentheses in Table 1. There was no age or sex that was more prone to develop neurologic complications.

**Neurologic disease** A definitely higher incidence of neurologic complications oc



Fig 4



Fig 5

Fig 4 Case 2 Arterial embolization or spasm of the basilar artery in a 34 year old man not present at the first or second injection. Moderate atherosclerotic disease of the carotid system apparent

Fig 5 Case 3 Basilar artery occlusion in a 34 year old woman with cerebrovascular disease. The first vertebral angiography was normal but was probably responsible for the development of quadriplegia. The second vertebral angiography 3 weeks later disclosed occlusion of the basilar artery beyond the origin of the anterior inferior cerebellar artery (arrow)

occurred in patients with cerebrovascular diseases (Table 2). Five of 9 patients were known to have neurologic signs considered to be due to cerebrovascular diseases. In most of these, atherosclerotic plaques or arterial occlusions were demonstrated angiographically. Two patients had infratentorial tumours with increased intracranial pressure. There was no significant angiographic abnormality in 2 patients who were examined for evaluation of seizures.

*Arterial anatomy and angiographic technique* Two of the 9 neurologic complications occurred when the posterior cerebral arteries received blood supply from the carotid system and the vertebrobasilar system was quite small. RP\ 062 catheters were used for the angiography in 4 of the patients and RP\ 045 catheters in 3 patients. Two sequelae developed following vertebral angiography via the brachial route. Since the use of smaller catheters predominated in a ratio of four to one it would appear that larger catheters were associated with more complications than the former. The complications in





Fig 4



Fig 5

Fig 4 Case 2 Arterial embolization or spasm of the basilar artery in a 34 year-old man not present at the first or second injection. Moderate atherosclerotic disease of the carotid stem apparent.

Fig 5 Case 3 Basilar artery occlusion in a 34 year-old woman with cerebrovascular disease. The first vertebral angiography was normal but was probably responsible for the development of quadriplegia. The second vertebral angiography 3 weeks later disclosed occlusion of the basilar artery beyond the origin of the anterior inferior cerebellar artery (arrow).

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**Table 2**  
*Neurologic disease and neurologic complications*

<i>Diagnosis</i>	<i>No of patients</i>	<i>No of complications</i>
Normal	188	2
Supratentorial tumour	79	0
Supratentorial vascular lesion	45	5
Intraorbital tumour	7	0
Infratentorial tumour	71	2
Infratentorial vascular lesion	18	0
Infratentorial anomaly	4	0
Extracranial lesion	18	0
Other lesion	10	0
Unsuccessful examination	17	0
Total	457	9

**Table 3**  
*Type of angiography and neurologic complications*

	<i>No of examinations</i>	<i>No of complications</i>
Vertebral angiography only	144	6
Vertebral and carotid angiography		
Right and left carotid	64	2
Right or left carotid	130	1
Carotid angiography only		
Right and left carotid	52	0
Right or left carotid	92	0
Unsuccessful examination		
Vertebral angiography	8	0
Carotid angiography	10	0
Total	500	9

vertebral angiography occurred more frequently when there was better reflux into the contralateral vertebral artery, suggesting that the speed of injection may be an important factor

*Types of examinations* All the neurologic complications in this series of 500 examinations were associated with vertebral angiography (Table 3). Six patients underwent left vertebral angiography and 2 patients were examined by vertebral and right common carotid angiography while left vertebral and bi-

lateral carotid angiography was performed in one patient. There were no complications following internal, external or common carotid angiography. It must be remembered that the vertebral artery was outlined in 70 per cent of 500 examinations. The frequency of complications in the patients with filling of more than one vessel is no higher than that in those in whom only one vessel was outlined. Furthermore, there appears to be no relationship between the occurrence of sequelae and the amount of contrast medium or the number of injections.

### Discussion

A brief review must be made on complications reported in some other larger series in which a modern contrast medium was used in order to compare those due to the catheter technique with those of other methods. As regards vertebral angiography, LESTER & KLEE (1965) reported 7 neurologic complications in 337 examinations by a direct percutaneous technique including two fatal terminations. RUGGIERO et coll (1958), in a series of 277 examinations performed with a similar technique, experienced 9 sequelae, two of which were fatal. Other authors also had a similar incidence (MOVES 1961; SCATLIFR et coll 1965). The rate of changes due to vertebral filling following a retrograde brachial injection technique appears to be lower (GOULD et coll 1955; TATELMAN & SHEEHAN 1962). On the other hand, TAKAHASHI et coll (1969) reported an incidence of 8 neurologic complications in 250 examinations in which a catheter technique via the femoral or axillary artery was employed. NEWTON's series (1966) of 170 examinations by a catheter technique included 9 neurologic sequelae. Neither of the latter two authors reported fatalities.

The complications of carotid angiography by a direct puncture technique have been reported to be from 2.1 to 5.5 per cent (EIKEN & GORMSEN 1962; FEILD et coll 1962; PERRET 1966; SCHEINBERG & ZUNER 1963; PATTERSON & DUNN 1964). Changes due to carotid angiography by percutaneous femoral catheterization have however not been evaluated in a large series. NEWTON & GOODING (1968) reported no complications in their series of 76 cerebral angiograms in the pediatric age group. The incidence of neurologic sequelae in the present series by the catheter technique for carotid angiography appears to be considerably lower. Asymptomatic changes such as arterial spasm or embolization do occur again requiring careful application of the catheter technique to the carotid arteries.

The experience gained in 500 catheter cerebral angiographies suggests that the most important factor in the occurrence of neurologic signs is the speed of

the contrast medium injected per cross sectional area of the blood vessel. This concept may be exemplified by the fact that more changes occurred in the series when contrast medium was injected with larger catheters and when there was more reflux into the contralateral vertebral artery. The observation that the sequelae occurred more frequently in vertebral than in carotid angiography and in the small vertebrobasilar systems may also be explained by this premise. Because of this it is felt that the catheters for cerebral angiography, especially for vertebral angiography, should be smaller and the speed of the injection should be adjusted when larger catheters are used. Furthermore the speed of injection should be slowed when the posterior cerebral artery is supplied by the carotid system or the vertebrobasilar system is extremely small.

CRONGVIST *et coll* (1970) reported 7 patients with cerebral embolization as a major complication of percutaneous femoral catheterization. Emboli may theoretically be more frequent with the catheter technique. Two such incidents were encountered in the present series without evidence of further neurologic defects. The source of the embolus is considered to be blood clots formed in the catheters, or air emboli rather than dislodged atherosclerotic plaques. Because of this, repeated flushing of the catheters is necessary with heparinized saline with two syringes; this is important since blood clots may easily form in the syringes and be injected into the vessels when only one syringe is used for flushing. The low incidence of embolization in the present series is probably attributable to these precautions. CRONGVIST *et coll* (1970) emphasized that the catheter surface should be smooth and the examining time as short as possible to prevent thrombus formation around or inside the catheters. Another source of emboli may be atherosclerotic plaques dislodged by the catheter from the arterial walls. Although the latter may not frequently occur, flexible catheters should be used in the most gentle fashion.

Two instances of quadriplegia have been reported with vertebral angiography by percutaneous femoral catheterization (HOWIESON & MEGISON 1969; TAKAHASHI *et coll* 1969) although such severe complications are infrequent with the catheter technique. One patient had quadriplegia following percutaneous femoral catheterization. Permanent complications secondary to spinal cord or medullary injury have been reported with the direct puncture technique (SLGAR *et coll* 1949; SUTTON & HOARE 1951; EDERLI *et coll* 1962) and the incidence appears to be slightly higher than with the catheter technique.

The incidence of neurologic complications is increased in patients with cerebrovascular diseases in the present material. PATTERSON & DUNNING (1964) reported neurologic sequelae in 11 per cent of such patients, serious in 3.4 per cent. FEILD *et coll.* (1962) published severe or fatal complications in 0.9 per cent of 760 patients with cerebrovascular disease. All the four severe incidents

in the series of LESTER & KLEE (1965) occurred in patients with vascular insufficiency. Additional reports which describe a higher incidence in this particular condition also exist (SEDTIMIR 1955, SCHEINBERG & ZUNKER 1963, LEIKIN & GORMSEN 1962, PRIEBUM 1965, PERRET 1966). It may be concluded from these data that atheromatous disease of the cerebral arteries increases the complications of cerebral angiography. All the evidence suggests therefore that stricter indications for cerebral angiography should be applied in the examination of patients with cerebrovascular conditions in order to lower the incidence of neurologic complications.

There have been reports that the less experienced operators cause more changes than the experienced examiners (LESTER & KLEE 1965, SCATLIFF et coll. 1965). All the examinations in the present series were performed under close supervision by experienced neuroradiologists and no causal relationship could be obtained in this connection.

### SUMMARY

Complications in 500 cerebral angiographies performed by percutaneous femoral catheterization are described. These occurred in proportion to the speed of injection per cross sectional area of the vessels requiring smaller catheters and adjustment of injection pressure when the artery was small. It is also emphasized that patients with cerebrovascular diseases subjected to angiography must be examined with care.

### ZUSAMMENFASSUNG

Die Komplikationen bei 500 cerebralen Angiographien die mit perkutaner femoraler Katheterisierung durchgeführt wurde werden beschrieben. Diese traten im Verhältnis zur Geschwindigkeit der Injektion per Querschnittsfläche des Gefäßes auf wobei kleinere Katheter und Anpassung des Injektionsdrucks daran notwendig sind wenn die Arterie klein war. Es wird weiter hervorgehoben dass Patienten mit cerebrovaskulären Erkrankungen die einer Angiographie unterzogen werden vorsichtig untersucht werden müssen.

### RÉSUMÉ

Les auteurs décrivent les complications de 500 angiographies cérébrales faites par cathétérisme fémoral percutané. La fréquence de ces complications est proportionnelle à la vitesse d'injection rapportée à la surface de section transversale des vaisseaux qui nécessite des cathéters plus fins et une adaptation de la pression d'injection quand l'artère est petite. Les auteurs insistent aussi sur la nécessité de faire l'examen angiographique prudemment chez les sujets qui sont atteints d'affections cerebrovasculaires.



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## HEART RATE REACTIONS DURING CEREBRAL ANGIOGRAPHY

The value of ECG recordings

by

GUNNAR TÖRNELL

Untoward effects and complications at angiography appear to be caused less frequently nowadays by the contrast medium. A change seems to have occurred when contrast media of the iodopyracete and acetrizoate types were abandoned and those of the diatrizoate and later metrizoate and iothalamate variety were introduced. The complications arising from puncturing of the artery or the catheterization seem to be more frequent than those produced by the contrast medium. Every factor must, however, be considered.

It has long been known that changes in cardiac rhythm and blood pressure may occur during carotid angiography (WATHIERALL 1942, GREITZ 1956, TÖRNELL 1963). During the first seconds after the injection bradycardia is frequently recorded and may cause a decrease in blood pressure. Tachycardia may be recorded ten to thirty seconds after the injection. These cardiovascular effects may be severe with asystolia for seconds when acetrizoate is used. Contrast media of the diatrizoate, metrizoate and iothalamate type produce only slight cardiovascular changes. In patients with a normal cerebral circulation are rarely

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Table 1

Bradycardia expressed as the mean deviation of heart cycle in hundredths of a second and in whom time in seconds on intracarotid injections of the contrast media of methylglucamine salts of diatrizoate, iothalamate and metrizoate.

	Diatrizoate M 100		Iothalamate M 100		Metrizoate M 100	
	60		60		60	
	20-30 ml		20-30 ml		20-30 ml	
	Brady	Injection	Brady	Injection	Brady	Injection
	cardia	time	cardia	time	cardia	time
Range	0-3	1.9-6.4	0-30	1.12-2.00	0-3	1.16-2.03
Mean	3.8	1.33	4.6	1.32	4.5	1.33
Per cent of cases with bradycardia more than 0.04 s	1		2		30	

\* Special prepared for this investigation

patients referred for carotid angiography. Those examined under general anaesthesia and those with cardiac arrhythmias were not included. Pethidine and atropine were used for pre medication. The amount of pethidine varied but the atropine dose was always 0.5 mg. The contrast medium was injected with an automatic syringe through a percutaneous needle into the internal or common carotid artery. The ECG roentgen exposures and injection time were recorded in all instances. The patients were selected and the investigation was performed as described in 1967 GRETZ & TÖRNELL. The following contrast media were used for all the patients: methylglucamine diatrizoate 60% (Angiografine), methylglucamine iothalamate 60% (Conray) and methylglucamine metrizoate 60.5% (Iopaque). The content of iodine was 232, 282 and 280 mg/ml, respectively. 5 ml of the three different contrast media were used at each injection with the same position of the needle. The order in which the contrast media were injected was randomized. Statistical analysis was performed.

The degree of bradycardia was expressed as the difference in hundredths of a second between the longest heart cycles after and immediately before the injection. The tachycardia was measured as the difference between the shortest cycles before and after the injection. The five beats before injection were measured. Bradycardia usually occurred within ten seconds after the start of the injection. Tachycardia was often observed after ten seconds. The ECG was recorded for 30 seconds after the end of the injection.

In order to obtain more information on the question as to where bradycardial reaction is more often elicited such reactions were compared in another 12

Table 2

*Bradycardia expressed as maximal prolongation of heart cycle in hundredths of a second after selective injection of contrast medium into the external carotid artery and either the internal or common carotid artery of the same side and in the same patient*

	Common carotid artery	Internal carotid artery	Internal carotid artery
Range	0-38	0-20	0-10
Mean bradycardial reaction in 22 patients	18	12	15
	49		
Per cent of cases with bradycardia more than 0.04 s	32		0

patients after injection into the external and internal carotid arteries in the same patient and on the same side, the reactions were also compared after injection into both the external and common carotid arteries in a further 10 patients. The selectivity of the injection was controlled in all cases simultaneously with the LCG recording. The bradycardial reaction from the injection into the vertebral artery was compared in 9 other patients with that after injection into the internal or common carotid artery. The injections both into the carotid and the vertebral arteries were performed by direct needle puncture. Patients in whom the injections were made through a catheter were excluded from the present material.

## Results

A comparison of the reactions of the contrast media in the first 30 patients appears in Table 1. The bradycardial reaction of the three contrast media did not differ much, the mean of diatrizoate being 3.8, iothalamate 4.0 and metrizoate 4.5 hundredths of a second. The statistical analysis revealed no significant difference. The injection times were measured, the mean injection time for diatrizoate being 1.55, iothalamate 1.52 and metrizoate 1.58 s.

For comparison of the reactions elicited after injection into the external carotid artery with those into the internal or common carotid artery, the injections were controlled by serial angiography performed simultaneously with the LCG recording in the other 22 patients. Selective injections into the external as well as the internal carotid artery without leakage to the external carotid artery were obtained in 12 of the patients. In the other 10 patients, attempted internal carotid injections were tainted by overflow into the external carotid artery, these were registered as injections into the common carotid artery. The mean brady

Table 3

*Bradycardia or tachycardia expressed as maximal variation of heart cycle in hundredths of a second after selective injection of contrast medium into the vertebral artery and either the internal or common carotid artery in the same patient*

	Vertebral artery		Internal or common carotid artery	
	Brady cardia	Tachy cardia	Brady cardia	Tachy cardia
Range	0-40	0-6	0-54	0-70
Mean reaction in 9 patients (= change of heart cycle in 0.01 s)	13.6	1.4	22.6	4.2
Cases with a variation more than 0.04 s	5	1	7	3

cardiac reaction in the common or internal carotid artery was 4.9 (that is a prolongation of the heart cycle by 0.049 s) a reaction three times more than that in the external carotid artery of the same patients the mean reaction being 1.5 (Table 2). Thirty-two per cent of the 22 patients with an injection into the common or internal carotid artery had an increase in the cardiac cycle of more than 0.04 s against 9 per cent after selective injection into the external carotid artery.

Ten injections were made in 9 patients into both the vertebral and either the internal or the common carotid artery of the same patient and the heart rate was recorded (Table 3). The mean bradycardial reaction in the vertebral artery was 13.6 hundredths of a second and after injection into the common or internal carotid artery 22.6. The tachycardial reaction was slight and less after injection into the vertebral than into the common carotid artery. Bradycardia with a reaction more than 4 (= prolongation by 0.04 s of the heart cycle) was encountered less often: 16 after 5 out of 10 injections into the vertebral artery and after 7 out of 10 injections into the carotid artery.

No complications occurred in these series of patients. Those with cardiac arrhythmia were excluded from the material. Catheter injections were also excluded since they caused much stronger bradycardial reactions. Three patients who received four injections through a catheter into the vertebral artery had prolongation of the cardiac cycle by 0.026, 0.024 and 0.020 s respectively, and three catheter injections into the internal carotid artery caused an increase in the cardiac cycle by 0.022 and 0.18 s, respectively. The tip of the catheter was placed relatively high up in the vessel injected in all these patients.

## SUMMARY

The bradycardial reactions during cerebral angiography have been used to compare the pure methylglucamine salt of the three most used contrast media Urografin Isopaque and Conray. The reactions were larger and more frequent after injections into the cerebral vessels than into the extra cerebral vessels. The value of ECG recording during cerebral angiography is stressed.

## ZUSAMMENFASSUNG

Die Bradykardie Reaktionen während der cerebralen Angiographie wurden verwendet um das reine Methylglucamine Salz der drei am meisten verwendeten Kontrastmittel Urografin Isopaque und Conray zu vergleichen. Die Reaktionen waren nach Injektion in die cerebralen Gefäße stärker ausgeprägt und häufiger als nach Injektion in die extracerebralen Gefäße. Der Wert von ECG Registrierungen während der cerebralen Angiographie wird hervorgehoben.

## RÉSUMÉ

Les reactions bradycardiques au cours de l'angiographie cerebrale ont servi a comparer le sel pur de methylglucamine des trois moyens de contraste les plus utilises Urografin Isopaque et Conray. Les reactions sont plus importantes et plus frequentes apres injection dans les vaisseaux cerebraux que dans les vaisseaux extra cerebraux. L'auteur insiste sur l'utilite de l'enregistrement electrocardiographique pendant l'angiographie cerebrale.

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## ASYMÉTRIE DE LA FACE ET DES ARTÈRES VERTÉBRALES

A propos d'un cas de persistance unilatérale de l'artère hypoglosse

par

A. WACKENHEIM

L'asymétrie faciale est tellement fréquente qu'on a tendance à négliger sa signification médicale. Le crâne et la face sont des organes symétriques soumis à des phénomènes asymétriques. C'est ainsi que la latéralisation neurologique vient chez le droitier par exemple, solliciter plus fortement le côté droit grâce aux attaches musculaires de la base du crâne. Un deuxième facteur non négligeable est en rapport avec la disposition asymétrique des vaisseaux du cou. Dans ce travail, nous tenons à insister sur la fréquence de l'asymétrie faciale chez les malades qui consultent pour insuffisance vertébro-basilaire. Il est bien établi à l'heure actuelle que l'insuffisance vertébro-basilaire est favorisée par des dispositions anormales des artères vertébrales. Dans ce cadre nous attachons, en pratique, une importance clinique à l'asymétrie faciale qui, chez un sujet souffrant d'insuffisance vertébro-basilaire, est un argument en faveur d'une disposition asymétrique des artères vertébrales. Ces malades, dont un exemple est rapporté dans les figures 1 et 2, ont un tableau comprenant trois ordres de signes : (1) une symptomatologie clinique d'insuffisance vertébro-basilaire, (2) une asymétrie de la face, et (3) une disposition asymétrique des artères vertébrales (par exemple hypoplasie ou agénésie unilatérale, défaut d'aboutissement avec la vertébrale opposée, vertébrale borgne).



Fig 1 Hypoplasie de la face limitée à la région sous orbitaire gauche (type VI de notre classification)



Fig 2 Hypoplasie de l'artère vertébrale gauche chez le malade représenté sur la fig 1

Nous rapportons ici l'observation d'un tel malade son asymétrie vasculaire étant une anastomose « vertébro-carotidienne unilatérale par persistance de l'artère hypoglossale droite



Fig 3 Scoliose cranio-faciale dextro convexe chez le malade dont l'angiographie est illustrée sur la fig 4



a



b



c

Fig 4 Persistance de l'artère hypoglosse droite sous forme d'une anastomose entre l'artère vertébrale et l'artère carotide a) Sur les premiers temps arteriels l'anastomose n'est pas visible b) et c) L'anastomose (→) apparaît nettement opacifiée dans le sens vertebro-carotidien sur les clichés plus tardifs

Homme âgé de 44 ans se plaint d'une symptomatologie cochléo-vestibulaire droite de type Ménière et de petits signes pyramidaux irritatifs diffus sans valeur localisatrice.

À gauche il existe une discrète surdité de perception. On note d'autre part une scoliose crânio faciale dextro convexe (Fig. 3).

L'angiographie vertébrale révèle à droite la persistance d'une artère hypoglosse qui s'opacifie à partir de l'artère vertébrale (Fig. 4).

Cette observation vient illustrer par une variante rare l'asymétrie vasculaire chez un sujet atteint par ailleurs d'asymétrie faciale. L'asymétrie faciale que présentent ces malades peut adopter différents types, mais plus spécialement les types III et VI de la classification que nous avons proposée en 1969 (WACKENHEIM). Dans l'observation présente, il s'agit du type III ou scoliose crânio faciale.

## RÉSUMÉ

L'auteur insiste sur la fréquente association « signes cliniques d'insuffisance vertébro-basilaire — asymétrie faciale — disposition asymétrique des artères vertébrales ». Il rapporte l'observation d'un tel malade chez lequel l'asymétrie des artères vertébrales est la persistance unilatérale d'une artère hypoglosse.

## SUMMARY

The author lays stress on the frequent association of clinical signs of vertebro-basilar insufficiency — facial asymmetry — asymmetric disposition of the vertebral arteries. He reports a case of such a patient in whom the arterial asymmetry is the unilateral persistence of a hypoglossal artery.

## ZUSAMMENFASSUNG

Der Autor beschreibt die oft bestehende Assoziation von klinische Symptome einer vertebrobasilarer Insuffizienz Gesichtssymmetrie — asymmetrische Anlage der Arteria vertebralis. Es wird ein Fall beschrieben in dem die Vertebralis Asymmetrie eine einseitige Persistenz der Arteria hypoglossi war.

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## SUBEPENDYMAL VENOUS FILLING SEQUENCE AT CEREBRAL ANGIOGRAPHY

Influence of grey and white matter distribution

by

EUGENE WHITE and TORGVY GREITZ

A late filling of the septal vein in relation to other subependymal veins has been previously noted at cerebral angiography (GREITZ 1956) but no explanation of this phenomenon has been given. In principle the time of appearance of contrast medium in any given vein could be dependent on two factors. The length of the path the blood has to travel to reach the vein and the resistance to the blood flow through the area drained by the vein. The first principle has already been shown to be valid for the superficial veins (GREITZ & REUTER 1966). With regard to the second principle it is of interest that cerebral blood flow examinations using the isotope clearance method (LASSEN & INGVAR 1963, KETY 1965) have shown that the clearance curves obtained in cerebral blood flow measurements are consistent with a two compartment system with a fast phase representing flow in grey matter and a slow phase attributed to white matter flow. Therefore, it became of interest to analyze more carefully the drainage areas and the sequence of venous filling of the deep cerebral veins to discover if filling times could be correlated with the grey and white matter composition of the drainage areas.

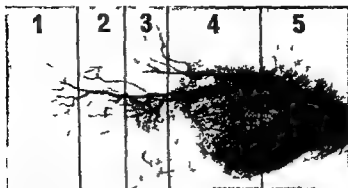


Fig 1 Autopsy material Selective blocking of medial longitudinal caudate vein by steel ball Coronal sections were made as indicated in lateral view

**Material and Methods** Ten brains from autopsy material of less than 24 hours were carefully removed together with the tentorium vein of Galen intact. After a coronal section through the midbrain at the level of the colliculi the vein of Galen and the internal cerebral veins were carefully exposed and a 0.048 mm OD catheter was introduced under fluoroscopy into either the internal cerebral or basilar vein and secured with a ligature. Alternatively a 0.7 mm steel bearing could be posed into position blocking a small tributary in order to delineate the drainage areas of adjacent veins. A 10% micropaque suspension was injected at the rate of 2 ml per minute up to 100 ml using a mechanical pump. In order to prevent leakage of contrast medium during fixation and subsequent roentgen examination 20 ml of micropaque gelatin suspension were injected for sealing. The fixed specimens were sectioned and photographed in order to demonstrate the selected area of the particular specimen (Figs 1-3). Information in regard to the drainage areas was sought in the literature (SCHLESINGER 1939, JOHANSSON 1954, FERNER 1958, KAPLAN 1959, HUANG & WOLF 1963, 1964, HASSLER 1966, GILLILAN 1968, STEPHENS & STILWELL 1969) and particular attention was paid to those areas inadequately covered or subject to disagreement.

In order to analyze the normal sequence of filling of the subependymal veins 26 angiographies from patients without any clinical or radiologic signs of a focal intracranial lesion were reviewed. This material included mainly patients without neurologic deficits having either non focal epilepsy or headache. The age of the patients ranged from 25 to 56 years with a mean age of 36 years. The commencement, the maximum and the termination of filling

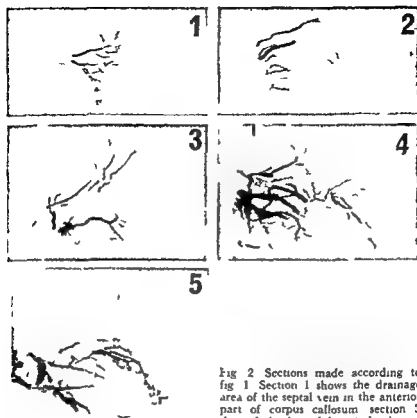


Fig 2 Sections made according to fig 1 Section 1 shows the drainage area of the septal vein in the anterior part of corpus callosum section 3 that of the lateral longitudinal caudate

were recorded with regard to the thalamostriate internal cerebral and basilar veins and their tributaries including the septal the medial and longitudinal caudate posterior terminal direct lateral and medial atrial veins The hippocampal the inferior ventricular and lateral atrial tributaries to the basilar vein were also included Other veins not belonging to the subependymal system but included in the investigation were the insular veins the inferior striate veins as well as the posterior pericallosal vein Filling times were expressed in seconds related to the maximum filling of the carotid siphon and to the beginning of the internal cerebral veins The commencement of filling of every individual vein was also related to every other individual vein as either preceding simultaneous or following A colour subtraction technique similar to that of LILIEQUIST & WELANDER (1969) was found to be useful for demonstrating the different stages of venous filling in a simultaneous image (Fig 13)

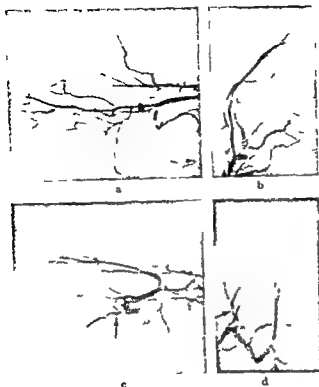


Fig 3 Autopsy material a) and c) Lateral views b) and d) Axial views a b) Selective filling of the drainage area of the septal vein by blocking of adjacent vein c d) Selective non filling of the septal vein reveals distribution of inferior portion (not described earlier) of medial longitudinal caudate vein

## Results

**Drainage areas** It was found that the subependymal veins could be divided into 3 types. Those draining only white matter those draining only grey matter and those draining both (Fig 6). Included in the latter group are for obvious reasons the main collecting channels such as the internal cerebral vein and the basilar vein receiving the different types of draining veins. The septal vein in agreement with HASSLER and STEPHENS & STILLWELL is a pure white matter draining vein receiving blood from the white matter of the frontal horn including the corpus callosum. Other pure white draining veins are the medial atrial vein draining white substance adjacent to the medial walls and the roof of the atrium (Fig 4) the latter being the corpus callosum the lateral atrial veins draining the lateral wall of the atrium the inferior ventricular vein draining the superior and lateral wall of the temporal horn i.e. the tapetum of the corpus callosum and the posterior septal veins emerging from the roof of the body of the lateral ventricles. The lateral extension of this part of the corpus callosum is drained by the trans-cerebral veins (KAPLAN





Fig 4 Autopsy material Lateral view (top) and axial view (bottom) The medial atrial vein drains only white matter



Fig 5 Autopsy material Axial view The posterior terminal vein (→) receives no tributaries from the thalamus located medially to this vein

1959) which empty into the longitudinal caudate and the posterior terminal veins. Veins draining exclusively grey matter are the medial caudate veins, which drain the head of the caudate nucleus and which may appear as one or two trunks or several fine branches and veins draining into the thalamostriate and the internal cerebral veins. The inferior striate veins draining the basal ganglia are almost exclusively grey matter draining veins and empty into the foremost part of the basilar vein, which has no significant white matter contribution. In this investigation the insular veins and the posterior pericallosal (cerebral) veins were included and were found to be grey matter draining veins, the posterior pericallosal vein receiving tributaries from the cortex of the surrounding hemisphere. Apart from the already mentioned main collecting

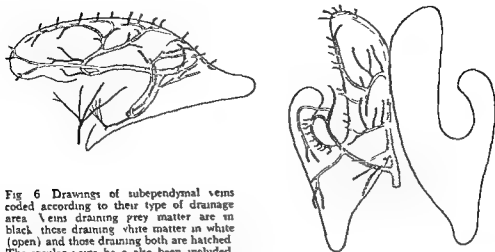


Fig 6 Drawings of subependymal veins coded according to their type of drainage area. Veins draining grey matter are in black, those draining white matter in white (open) and those draining both are hatched. The insular veins have also been included.

channels such as the internal cerebral vein of Galen and the posterior part of the basilar vein, blood from both grey and white matter is carried through the longitudinal caudate, the posterior terminal and the thalamostriate and the hippocampal veins. The thalamostriate, the internal cerebral and the posterior part of the basilar veins are all receiving short tributaries from the adjacent grey matter, i.e. the caudate nucleus and the thalamus. The direct lateral vein, when it occurs, is draining both grey and white matter. We have found that the longitudinal caudate vein varies in the extent of tributaries from the caudate nucleus and may actually anastomose with the medial caudate (Figs 11-14).

*Sequence of filling.* A definite correlation between the appearance time of the different subependymal veins and the type of drainage area was found. When arranged in the order of their appearance time, the grey matter draining veins appear first, the veins carrying mixed blood second and the white matter draining veins last (Figs 7, 8, 10, 11). It is interesting that there also appears to be a correlation between the duration of filling and the type of drainage areas, that is, the grey matter draining veins have a short duration of filling, the white matter draining veins a longer period and the mixed have the longest duration of filling (Figs 8, 9). There appears also to be a time gradient among the veins of a particular type, suggesting influence by the length of the path travelled by the contrast medium on the arterial side before the drainage area and the drainage vein, e.g. in the grey matter draining group, the posterior pericallosal usually filled after the inferior striate veins and in the white matter draining group, the medial atrial vein always filled after the septal vein (Fig 11).

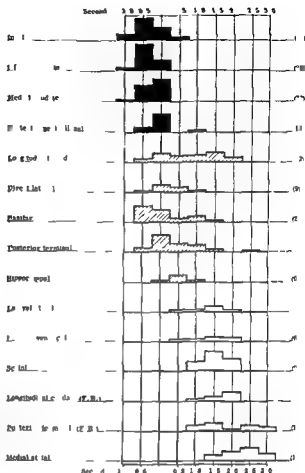
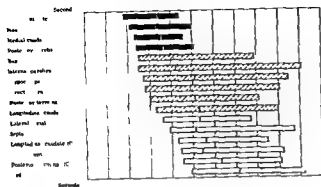


Fig 7 Diagram showing commencement of filling of deep veins in relation to that of internal cerebral vein in 26 normal subjects. The parenthesized numbers to the right represent total number of veins recorded which does not total 26 because of non filling or overlap preventing identification. The veins are indicated as black, white or hatched bars in accordance with symbols in Fig 6. F II refers to fine branches i.e. white matter draining contributions.

### Discussion

The classification of the subependymal veins into three groups i.e. those draining grey matter, those draining white matter and a mixed group represents a restatement of anatomic information established over the years by many contributors. SCHLESINGER (1939) distinguished and named the medial longitudinal caudate system and pointed out the grey matter drainage of the former as distinct from the mixed drainage of the latter. The work of BROWNING (1884) referred to by HUANG & WOLF in which the inferior ventricular vein was described and named, could unfortunately not be obtained. Different authors have not agreed upon certain drainage areas. FERNER (1958) claimed that the posterior terminal received contribution from the thalamus but this has not been confirmed by our findings (Fig 5). In agreement with



**Fig 1** Time of appearance maximum filling and disappearance in seconds related to maximum filling of the carotid siphon. Average values

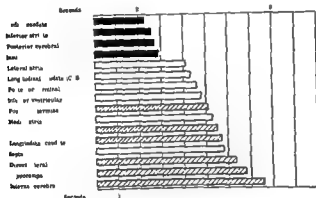


Fig 9. Length of venous phase expressed as the average total filling time in seconds.

STEPHEN & STILWELL we have found the thalamus to be drained mostly directly into the internal cerebral vein except for a small contribution from the posterior thalamus to the basilar vein. This contribution becomes significant with a long anterior choroid extending up behind the pulvinar of the thalamus. When this occurs the time lag between the filling of the anterior and posterior part of the basilar vein may diminish or disappear. The posterior terminal vein drains the body of the caudate and also the adjacent white matter and the superior aspect of the lentiform nuclei. Again, in agreement with STEPHENS & STILWELL we have found that the direct lateral when present, probably drains both the thalamus and caudate as well as adjacent white matter. As mentioned, the white matter contribution to the inferior striate veins and the anterior portion of the basilar vein is very small in comparison to the grey matter contribution. This implies that from an angiographic point of view these veins may be regarded as pure grey matter draining veins. Similarly the contribution from the grey matter of the tail of the caudate nucleus to inferior ventricular veins.

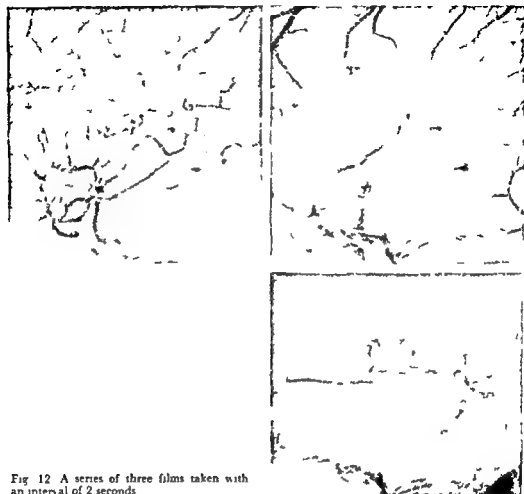


Fig. 12 A series of three films taken with an interval of 2 seconds

REUTER 1966) For example, the area drained by the septal vein is supplied by the most lateral branches of the lenticulostriate arteries (GILLILAN 1968 VAN DEN BERGH 1969) which represents a shorter path than that leading to the area drained by the medial atrial veins which area is identical to the posterior part of the corpus callosum, supplied by the distal branches of the pericallosal artery. Since a drainage area has a limited arterial supply, an abnormal draining vein can also imply possible abnormality in a limited number of arteries. This re-emphasizes the need for information concerning the arterial supply for the particular venous drainage area. Such an analysis may be of value in detecting arterial disease as well as in the diagnosis of pathology arising in the drainage area such as tumor or inflammatory processes (Fig. 15)

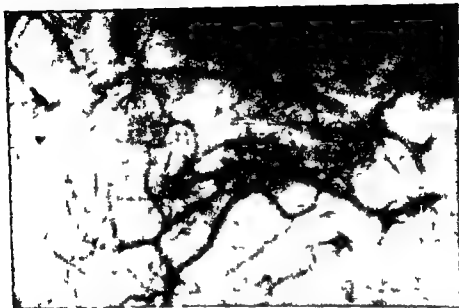


Fig 13 The series of films in fig 12 has been summarized to demonstrate the use of colour subtraction technique for the identification of the time relationship between filling of different veins. The image is built up by three colours: magenta, yellow and cyan, in that order, representing the time sequence. The septal and medial atrial veins, which drain white matter and fill late, are in blue; the medial caudate, which drains grey matter, is in red.

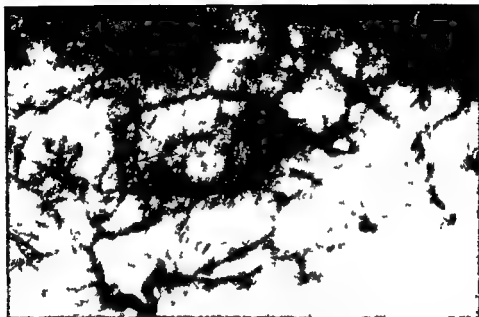


Fig 14 Colour subtraction Normal case As an anatomic variation a venous arcade exists between the medial caudate and the longitudinal caudate veins the t<sub>1</sub>m caudate veins appearing simultaneously (in red) with several intervening branches between them The tributaries from the roof i.e from which matter are appearing late (in blue) after the medial caudate and the arcade connections



Fig 15 Astrocytoma in the basal ganglia. Early drainage of the septal vein ( $\rightarrow$ ) indicates involvement of the lateral anterior corner of the corpus callosum. An early draining insular vein ( $\rightarrow$ ) suggests extension of tumor to the Sylvian fossa.

### Conclusions

The normal sequence of filling of the subependymal veins reflects primarily grey and white matter composition in the drainage areas and secondarily, with regard to veins with identical types of drainage areas the length of the path the blood has to travel on the arterial side to reach the drainage area.

Variations in grey and white composition of drainage areas affect the appearance time of a particular variety of a vein.

Knowledge of the normal sequence of filling may be utilized in the early diagnosis of various pathologic conditions.



The filling sequence of the independent veins can thus be a proof that the fast and slow components of the clearance curve, observed at cerebral blood flow measurements, are related to the anatomical structures, their function being drainage and absorption.

## SUMMARY

The drainage rates of the independent veins have been measured by a selective cannulation technique in awake sheep preparations. The distribution of fast and slow rates in the drainage rates of these veins is related to the time of venous filling at arteriography which proves the venous flow dependence of the independent venous drainage to the brain.

## ZUSAMMENFASSUNG

Die Abflussgeschwindigkeit der unabhängigen Venen wurde mit Hilfe einer selektiven Injektion einer mit radioaktivem Phosphor versehenen Farbstofflösung, die die Verteilung der Drainageraten von Blasen aus den arteriographisch nachweisbaren Venen darstellt, gemessen. Die Abflussgeschwindigkeit der unabhängigen Venen ist von der Füllungszeit der unabhängigen Venen abhängig, was die Abflussabhängigkeit der unabhängigen Venen zum Gehirn beweist.

## RESUME

Les débits de drainage des veines indépendantes ont été mesurés au moyen d'une technique de cannulation sélective. La distribution de la mesure des débits de drainage dans les veines est en relation avec le temps de remplissage des veines à l'angiographie ce qui prouve la dépendance du débit des veines indépendantes du débit sanguin cérébral dépend de la seule structure drainée.

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## A PERCUTANEOUS BALLOON CATHETER TECHNIQUE FOR THE TREATMENT OF INTRACRANIAL ANEURYSMS

by

M H WHOLEY L KESSLER and M BOEHMKE

Balloon catheters of various types have been designed (CARLENS *et coll* 1951, HAVERLING 1970 NORDENSTROM 1954 1962 1966 STRAUBE & DOTTER 1963) The present authors have designed a series of balloon catheters intended to be used percutaneously for the control of hemorrhage Although their principle application has been within the visceral circulation recent extensions of the method demonstrate potential value for the control of certain intracranial aneurysms for carotid cavernous fistula, and possibly for temporary control of carotid circulation during intracranial surgery

Operative occlusion of the internal or common carotid artery has frequently been used in the treatment of certain intracranial aneurysms This may be done by immediate ligation or by a gradual occlusion requiring one or two open operative procedures The simplicity of intracranial occlusion by means of the balloon is similar to any other catheterization procedure with controlled observation of the balloon and tolerance of occlusion by the patient being continuously monitored

The initial method consisted of positioning an Ödman—Ledin catheter (ID 1.8 mm) within the internal carotid artery via a percutaneous femoral approach

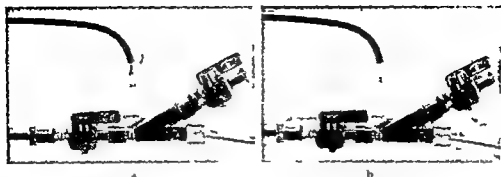


Fig 1 a) Unexpanded balloon catheter within a catheter. Proximal Y adaptor. Outer catheter has an ID of 1.8 mm. b) Expanded balloon diameter of 1.5 cm. Y adaptor allows flushing proximal to the balloon.

A polyvinyl balloon catheter was then passed through the catheter and positioned just distal to the catheter tip (Fig 1). Controlled expansion of the balloon with a contrast medium could be observed on the television monitor. If occlusion of the carotid was tolerated the balloon was left expanded until thrombosis occurred. In the three patients treated by this method thrombosis occurred in 24 to 48 hours (WHOLEY et coll 1970).

The polyvinyl catheter measures 1.3 mm OD with the balloon expanding to 1.5 cm. At the completion of the procedure the catheter system is withdrawn and pressure applied to the puncture site.

The original system required a Y adaptor on the proximal end of the catheter where sampling, flushing or observation of the balloon was possible. Unfortunately, observation distal to the expanded balloon was not possible. Consequently a double lumen polyvinyl catheter that could be inserted percutaneously over a 0.9 mm guide wire was constructed. External diameter of the catheter is 2.6 mm and balloon expansion occurs to 1.5 cm. A proximal luer lock seal allows prolonged distension of the balloon without leakage. Balloon position is either 0.5 cm or 3.0 cm from the tip and this is dependent on the examinations and curvatures necessary for the catheter (Fig 2).

Although vinyl can be preformed by heat and cooling to the solid state, it has an extremely limited memory and rapidly loses its form at body temperature. Consequently we have utilized a controllable guide for manipulation (Fig 3). More recently we have incorporated metallic wires within the wall for improved torque and curvature memory and have eliminated the necessity for controllable guides which in our experience have been generally unsatisfactory.

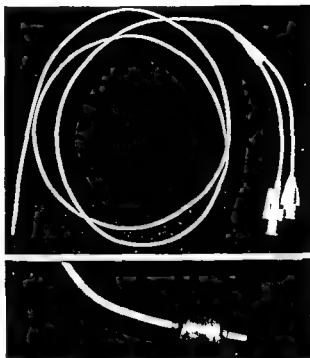


Fig 2 Upper view Newly designed double lumen polyvinyl catheter OD 2.6 mm Lower view Latex balloon diameter expanded to 1.5 cm

### Case reports

*Case 1* A 48 year-old female experienced sudden onset of headache and neck pain four days prior to admission. Examination revealed nuchal rigidity but no localizing neurologic signs. A spinal tap established the diagnosis of subarachnoid hemorrhage and angiography revealed a wide based vestigial aneurysm of the left internal carotid artery. The aneurysm was clipped successfully with a Mayfield temporary clip and the patient did well. Post operative angiography one week later showed the aneurysm to be patent with the clip dislodged. At reoperation the following day the aneurysm was wrapped with muscle. The patient was discharged in good condition.

Repeat angiography performed two years later showed the aneurysm to be substantially larger. With the patient under mild sedation a percutaneous balloon catheter was directed into the left internal carotid artery under televised fluoroscopy. The balloon was gradually expanded until the artery was totally occluded. The patient tolerated the total occlusion. Routine clamp monitoring procedures were instituted but proved unnecessary. Films of the neck were exposed at 12, 24 and 48 hours. After 48 hours angiography by femoral catheterization demonstrated a completely occluded left internal carotid artery with the balloon deflated. Four vessel angiography demonstrated good intracranial circulation. The aneurysm was not visible. The catheter was withdrawn uneventfully. The patient complained of neck and orbital pains for two or three days but showed no neurologic deficit and was discharged six days after admission. Follow up angiography three months later showed persistent occlusion of the parent internal carotid artery; the patient's condition remained excellent (Fig 4).

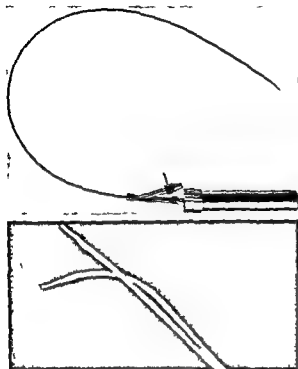
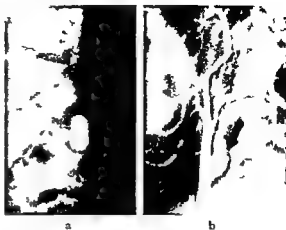


Fig 3 Upper view Guide wire (controllable) Lower view Flexion of catheter with guide wire in position (double exposure print)

**Case 2** A 58 year-old female experienced subarachnoid hemorrhage with only temporary loss of consciousness but without localizing neurologic signs. Angiography revealed a large bilobed aneurysm originating from the right internal carotid artery below the anterior clinoid—a risky challenge for intracranial clipping. Carotid ligation was considered the procedure of choice. The right common carotid was catheterized and the balloon catheter was advanced into the internal carotid artery and expanded. Unfortunately, the patient did not tolerate occlusion; despite prompt deflation of the balloon, left hemiplegia persisted and the patient's condition remained poor. The deflated balloon catheter and outer catheter were removed four days later (Fig 5).

**Case 3** A 67 year-old white male was admitted with massive bleeding from the right ear and both nostrils. Roentgenograms of the skull and facial bones demonstrated a fracture of the left frontal bone extending into the roof of the orbit. Another fracture was seen through the medial margin of the left orbit extending into the ethmoid sinuses. Fluid was noted in the frontal sinuses and maxillary antra indicating the presence of hemorrhage. The hematocrit at the time of admission was 43%; hemoglobin 14.2 g. These values dropped to a hematocrit of 30% and the hemoglobin to 10.6 g during the epistaxis. Three units of whole blood were given. The nose bleed was fairly well controlled with packing. However, rebleeding recurred on two occasions. About two weeks after the accident, occlusion of both external carotid arteries was obtained via balloon catheters. The balloon catheters were removed after 48 hours. There was no recurrence of the bleeding (Fig 6).

Fig. 4 a) Increasing aneurysm at junction of posterior communicating and internal carotid arteries treated by balloon occlusion of the internal carotid artery. The balloon was left in the expanded position for 48 hours. b) Thrombosis of internal carotid artery. The aneurysm did not fill from either the opposite carotid or vertebral injections and presumably was thrombosed.



### Discussion

The usual methods of carotid occlusion require operative exposure. Clamps are useful for flow control but are cumbersome and sometimes confusing; restoration of blood flow is not always achieved promptly by the inexperienced worker. Threats of wound infection, slough by a permanent clamp, and unsightly operative scarring are drawbacks of these techniques. The method described above provides a simplified approach to proximal arterial occlusion and lacks such drawbacks. The procedure is no more painful than angiography and can be performed on an unanesthetized patient; thus tolerance to occlusion can be observed and restoration of flow achieved easily without removing the dressings and without a confusing array of instruments protruding from the neck wound. Either gradual internal or common carotid occlusion can be elected with minimal manipulation.

Future use of this technique may include temporary proximal artery occlusion during intracranial surgery. The catheter or catheters could be inserted prior to surgery, being inflated only when necessary and with the aid of mobile televised fluoroscopy. This technique might also be used as a temporary procedure in active or threatened bleeding when emergency intracranial surgery appears to be unwise, facilitating prompt and definitive treatment without time-consuming preparation. The patient in Case 1 refused permission for another operation but accepted this procedure. Case 2 demonstrates an aneurysm which would be unsuited to intracranial clipping. Our failure cannot necessarily be attributed to the technique but rather is a common instance of cerebral infarction resulting from carotid ligation. Case 3 is an additional application of the method for external carotid control when surgery is not feasible.



Fig 5 (Left) Contrast medium in the carotid system with balloon occlusion too high to permit prompt washout from the proximal internal carotid

Fig 6 (Above) B lateral occlusion of both external carotid arteries. Balloon expanded with contrast medium. Both internal carotids are patent

A possible source of danger arises if early deflation of the balloon is necessary and the artery is not yet permanently thrombosed. Clot formation may have begun in a cul de sac proximal to the balloon and an embolus might be dislodged. Experimental proof of a stagnant stream has been demonstrated in the experimental animal and in humans by cine angiography when the cul de sac between the balloon and the parent artery is excessively long. This can be avoided by placing the balloon in close proximity to the carotid bifurcation so that rapid washout of contrast medium can be demonstrated.

### Addendum

Since the writing of this article three more patients with aneurysms of the internal carotid artery have been treated with occlusion of the internal carotid artery by means of a balloon catheter.

### SUMMARY

Balloon catheters that can be inserted percutaneously either as a primary catheter or through a parent catheter have been described as a possible alternative for the control of certain inaccessible intracranial aneurysms. Additional neuroradiologic applications include operative control of the carotid circulation during surgery, carotid cavernous fistula and external carotid control with either partial or total occlusion being entirely possible with essentially no more manipulation than a routine catheterization procedure.



## ZUSAMMENFASSUNG

Ballon Katheter die percutan entweder als Primärkatheter oder durch einen vorhandenen Katheter eingeführt werden können werden als mögliche Alternative zur Kontrolle gewisser unzugänglicher intracraneller Aneurysmen beschrieben. Zusätzliche neuroradiologische Anwendungen einschliesslich der operativen Kontrolle der Carotis Zirkulation in der Chirurgie der Kontrolle der cavernösen Carotidfistel und der Carotis externa mit entweder partieller oder totaler Occlusion sind vollständig ohne ein prinzipiell anderes Verfahren als eine routinemässige Katheterisierung möglich.

## RÉSUMÉ

L'introduction de cathétères à ballonnet qui peuvent être introduits par voie percutanée soit comme cathéter primaire soit par l'intermédiaire d'un cathéter principal est décrite comme une solution possible pour le traitement de certains anévrismes intracrâniens inaccessibles. D'autres applications neuroradiologiques sont l'arrêt peropératoire de la circulation carotidienne le traitement des fistules carotido-caverneuses et de la carotide externe avec la possibilité de réaliser une occlusion partielle ou totale des vaisseaux par une technique qui ne comporte pratiquement pas plus de manipulations qu'un cathétérisme ordinaire.

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## CEREBRAL DEATH



## DONNÉES RADIOLOGIQUES, ELECTRO ENCEPHALOGRAFIQUES ET ISOTOPIQUES DANS DES COMAS DÉPASSÉES

par

G ARTEL M AKERMAN F HERTZOG et CHANTAL BAMBERGER BOZO

Le diagnostic rigoureux de la « mort cérébrale » peut, bien entendu être posé sur des critères cliniques et électro-encéphalographiques qui ont le plus souvent suffisants pour donner une certitude. Mais la gravité de la décision qui s'ensuit (arrêt de l'assistance respiratoire et vaso-pressive) est telle que l'on est amené à chercher une confirmation de l'arrêt circulatoire par des explorations complémentaires tels que l'angiographie et les techniques radio-isotopiques.

*Matériel* Au cours des derniers 15 mois ont été explorés 12 malades présentant un état entrant dans la définition électro-clinique de « cerebral death ». Ces malades étaient arrivés à ce stade de mort cérébrale, soit par un accident cérébral aigu (rupture d'anévrisme par exemple), soit par un œdème cérébral irréversible consécutif à un arrêt cardiaque prolongé avec anoxie cérébrale. Enfin dans un cas la mort cérébrale représentait l'évolution terminale d'une encéphalite nécrosante subaiguë.

À titre comparatif ont été examinés 2 malades qui avaient présenté un arrêt cardiaque prolongé suivi de coma chronique pendant plusieurs mois dont un du type mutisme akinétique (FISCHGOLD & MATHIS).

**Tableau**  
*Matériel et résultats*

Clinique	N	FEG	Angiographie	Débit sanguin cérébral ( $^{131}\text{Xe}$ )	Scintigraphie ( $^{99}\text{Tc}^m$ )
« Mort cérébrale » par					
Accident cérébral aigu	9	Nul	Arrêt circulatoire	16 ml/min/100 g (1 cas)	Arrêt circulatoire (2 cas)
Anoxie cérébrale	2	Nul	Arrêt circulatoire		
Encéphalite subaigue	1	Nul	Arrêt circulatoire		Arrêt circulatoire
Comas chroniques	2	Déprimé	Normale	30 et 36 ml/min/ 100 g	

**Méthodes** Les enregistrements électro-encéphalographiques ont été réalisés en suivant les recommandations du « Brain Death Committee of the International Federation of Societies for EEG and Clinical Neurophysiology »

Les angiographies ont toutes été pratiquées par cathétérisme sélectif d'au moins trois des gros troncs artériels cérébraux (carotide primitive gauche et droite, vertébrale gauche). Dans trois cas la vertébrale droite a également été examinée.

Deux techniques radio-isotopiques ont été employées chez un certain nombre de malades. (1) La mesure du débit sanguin cérébral à partir des courbes de clearance du  $^{131}\text{Xe}$  injecté par voie carotidienne. Notre expérience de cette méthode repose essentiellement sur des études expérimentales chez le chat, après embolie gazeuse cérébrale, et sera publiée par ailleurs. (2) La scintigraphie avec une caméra à scintillations par injection intraveineuse de pertechnetate de  $^{99}\text{Tc}^m$ . Cette technique a été proposée récemment par GOODMAN et coll. Nous utilisons l'incidence frontale qui permet d'apprécier simultanément la circulation dans les deux hémisphères cérébraux.

### Résultats

Dans les 12 cas où l'EEG avait montré un trace nul, il a été constaté un arrêt circulatoire complet à la base du crâne à l'angiographie (voir Tableau).

Le débit sanguin cérébral mesuré dans un cas a été de 16 ml/min/100 g. Cette valeur relativement élevée est sans doute liée à un reflux de la solution de

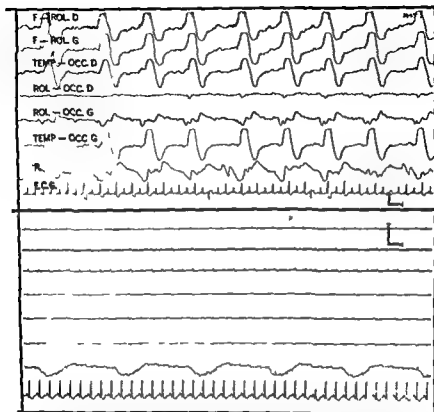


Fig 1 Traces électro-encéphalographiques à la phase terminale d'une encephalite necrosante subaigüe. En haut : Activité lente periodique stereotypée. En bas : Un jour apres au stade de la « mort cerebrale » trace nulle

venon dans le territoire carotidien externe bien que l'injection ait été faite par catheterisme selectif de la carotide interne

La scintigraphie intraveineuse au  $^{99}\text{Tc}^m$  realisee dans 3 cas a montre chaque fois d'une façon tres evidente un arrêt de la circulation intra-cranienne en concordance avec les resultats angiographiques

Chez le malade qui presentait une encephalite necrosante les explorations ont pu être repetees a deux reprises au cours de l'evolution terminale. Alors que le malade etait en coma stade III avec une EEG active lente periodique stereotypée (Fig 1) l'angiographie et la scintigraphie montraient une circulation cerebrale normale (Fig 2). Vingt-quatre heures apres, alors que l'electrogenese etait progressivement eteinte, la scintigraphie comme l'angiographie (Fig 3) ont apporte les memes resultats que dans les comas depasses a savoir un arrêt circulatoire complet a la base du crane

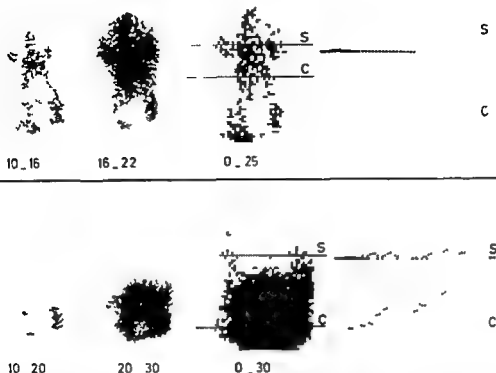


Fig 2 Meme cas Scintigraphie (camera a scintillations) apres injection intra veineuse de per technetate de  $^{99m}\text{Tc}$ . En haut Circulation intra-cranienne normale. En bas Un jour apres arret circulatoire. Les figures de droite representent les images digitalisees obtenues avec un analyseur a 4096 canaux et les profils d activite sur des coupes passant a la hauteur des arteres sylviennes (S) et carotidiennes (C).

Dans les deux cas de coma chronique ou l'electrogenese etait d'emblee detee norree profondement deprimee mais non nulle le debit sanguin cerebral etait de 30 ml/min/100 g et de 36 ml/min/100 g. L'angiographie selective a montre une circulation cerebrale normale d'apres les criteres angiographiques habituels.

### Conclusions

Dans notre experience le critere decisif de la « mort cerebrale » a pu être fourni par la constatation d'un arret circulatoire a l'exploration angiographique.

Cependant l'angiographie necessite le catheterisme selectif d'au moins trois des principaux troncs arteriels cerebraux. Il en est de meme pour la mesure du debit sanguin cerebral par injection intra arterielle de  $^{133}\text{Xe}$ . Dans cette derniere technique il est meme recommande de clamper les carotides externes, pour



Fig 3 Meme cas Exploration angiographique montrant l'arret circulatoire au niveau de la caroté interne et de la vertébrale gauches

éviter un reflux du venon dans les vaisseaux pericerebraux qui risque de fausser les mesures

Pour ces raisons la scintigraphie au pertechnetate de  $^{99}\text{Tc}^m$  nous paraît représenter une méthode de choix pour compléter le diagnostic clinique et électro-encéphalographique du « coma dépassé ». De réalisation simple et rapide, elle permet au moyen d'une simple injection intraveineuse de produit radio-actif de tester la circulation cérébrale simultanément au niveau des deux hémicrânes

## RÉSUMÉ

Une étude a été faite dans douze cas de « comas dépassés » et dans deux cas de comas chroniques en utilisant les méthodes angiographiques électro-encéphalographiques et isotopiques. L'étude angiographique a été pratiquée par cathétérisme sélectif des carotides internes et des vertébrales. L'étude isotopique a comporté la mesure du débit sanguin cérébral après injection carotidienne de  $^{133}\text{Xe}$  et l'angio-scintigraphie à la caméra à scintillations après injection intraveineuse de pertechnetate de  $^{99}\text{Tc}^m$ . Les auteurs montrent l'apport



de ces différentes méthodes qui témoignent de l'arrêt circulatoire cérébral irréversible. La scintigraphie isotopique à la caméra à scintillations leur parut du fait de sa simplicité un complément de choix du diagnostic clinique et électro-encéphalographique des « comas dépassés ».

## SUMMARY

An investigation has been performed in 12 cases of transient coma and 2 cases of established coma by means of angiography, electroencephalography and isotopes. The former was carried out by the selective catheterisation of the internal carotid and vertebral arteries. The isotopic examinations consisted in measuring the blood flow after the carotid injection of  $^{133}\text{Xe}$  and the angioscintigraphy was performed with a scintillation camera after the injection of  $^{99}\text{Tc}^m$ . The authors describe the value of these different methods in demonstrating irreversible arrest of the circulation. Isotopic scintigraphy with the scintigraphy camera would appear by reason of its simplicity to be the clinical method of choice in transient coma and electroencephalography the best method in transient coma.

## ZUSAMMENFASSUNG

Es wurden 12 Fällen mit einem vorübergehenden Koma und 2 Fällen mit einem ausgebliebenen Koma mittels Angiographie, Elektroenzephalographie und Isotopen untersucht. Die Angiographie wurde durch selektive Kathetersierung der Carotis interna und der Vertebralarterien vorgenommen. Die Isotopenuntersuchungen bestanden in Durchblutungsmessungen nach Injektion von  $^{133}\text{Xe}$  in die Carotis und eine Gefäßszintigraphie mit einer Szintigraphiekamera nach Injektion von  $^{99}\text{Tc}^m$ . Die Verfasser beschreiben den Wert dieser verschiedenen Methoden für den Nachweis eines irreversiblen Zirkulationstillstands. Die Isotopenszintigraphie mit der Szintigraphiekamera scheint wegen ihrer Einfachheit eine Methode der Wahl bei der klinischen und elektroenzephalographischen Diagnose beim vorübergehenden Koma zu sein.

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## KAROTIS- UND VERTEBRALISANGIOGRAPHIE BEIM HIRNTOD

VON

E. BUCHHEITZ UND C. KÄLFER

Unter dem Aspekt einer sinnvollen Reanimation und im Hinblick auf mögliche Organtransplantationen hat eine Umorientierung hinsichtlich der Todeszeitbestimmung stattgefunden. Die bisherige Konzeption richtete sich nach der Herz- und Atemtatigkeit. In letzter Zeit ist für bestimmte Situationen der irreversible Funktionsuntergang des Gehirns als entscheidendes Kriterium für den Eintritt des Todes in den Vordergrund getreten, trotz emotioneller Schwierigkeiten, die eine Todeszeiterklärung bei noch funktionierendem Kreislauf hervorrief. Aus den genannten Gründen ist jedoch die Gleichsetzung des Hirntodes mit der Todeszeitbestimmung an sich von großem praktischem Interesse. Der Nachweis einer absoluten Letalität aller Patienten mit zerebraler Zirkulationsunterbrechung trotz optimaler Reanimation läßt die künstliche Aufrechterhaltung von Herz- und Kreislauffunktion sinnlos erscheinen (KÄLFER & PENN, FOVVIS & FROWEIN). Eine vollständige zerebrale Zirkulationsunterbrechung führt in Abhängigkeit von der Zeit zu definierbaren Anoxieschäden. Der irreversible Funktionsuntergang des Zerebrums läßt sich definitiv bestätigen, wenn die intrakranielle Zirkulationsunterbrechung eine Zeitpanne die bei Beachtung eines Sicherheitsintervalles die Wiederbelebungszeit des Gehirns von 12–15

Minuten (GLEICHMANN et coll., SCHNEIDER) übersteigt, nachgewiesen werden kann

Die Schwierigkeit für die Bestimmung des zerebralen Todes liegt in seiner zweifel freien Verifizierung Anhand von klinischen Kriterien unter denen wir das tiefe Koma die zerebrale Areflexie und den Ausfall der Spontanatmung als obligat postulieren kann die Diagnose des Hirntodes nicht mit der erforderlichen Zuverlässigkeit gestellt werden Zur Absicherung der klinisch neurologischen Diagnose ist die Elektroenzephalographie unerlässlich geworden Allerdings weist ein Nulllinien EEG lediglich auf einen kortikalen Funktionsausfall hin und besagt nichts über Reversibilität oder Irreversibilität Daher spricht auch unter Berücksichtigung des klinischen Befundes ein über einen längeren Zeitraum nachweisbares und unter technisch standardisierten Bedingungen abgeleitetes Nulllinien EEG nicht absolut für einen zerebralen Funktionsuntergang Zudem sind in bestimmten Situationen — Intoxikationen anoxische Schädigungen — nach einem Nulllinien EEG hirnelektrische Restitutionen möglich so daß die Bedeutung des EEG in der Bestimmung des Hirntodes relativiert ist Es ist damit nicht überflüssig geworden Seine Bedeutung liegt vielmehr darin daß es die Veranlassung für eine den Hirntod beweisende zerebrale Angiographie bildet, d. h. in Verbindung mit der Klinik stellt ein Nulllinien EEG die Indikation zur Hirnangiographie dar Andererseits wird diese Untersuchung nicht vorgenommen wenn noch Reste einer hirnelektrischen Aktivität nachweisbar sind

Die Angiographie ist nach unserer Auffassung die entscheidende Untersuchungsmethode mit der eine sichere Diagnose des Hirntodes möglich ist Angiographischer Beweis eines irreversiblen Funktionsverlustes des Zerebrums ist ein vollständiger Zirkulationsstillstand in den intrazerebralen Arterien der durch eine Erhöhung des zerebrovaskulären Widerstandes hervorgerufen wird Für diesen ist eine intrakranielle Drucksteigerung infolge Hirnschwellung verantwortlich (STEINBEREITNER) Aus dem erhöhten Gefäßwiderstand resultiert zunächst eine reduzierte Hirndurchblutung wobei das Ausmaß von der Höhe des Schadelinnendruckes bestimmt wird Der intrakranielle Kreislaufstopp stellt somit das Endstadium dieser Entwicklung dar

Bisherige Beobachtungen einer intrakraniellen Kreislaufunterbrechung stammen aus neurochirurgischen Kollektiven mit Hirndrucksteigerungen bei Tumoren Blutungen oder Traumen des Gehirns und betreffen fast ausschließlich die Arteria carotis interna (ARONSON & SCATLIFF, FROEIN, GROS et coll., HEISMANEN, HORWITZ & DUNSMORE, HUNT et coll., KRAYENBUHL & YASARGIL, LANGFITT & KASSELL, MITCHELL et coll., NEWTON & COLGH, OKAY, PRIEBRAVI, RIINHEDE & ETHILBERG, TONNIS & FROEIN, TONNIS & SCHIEFER, WAPPEN, SCHMIDT) wogegen BUCHELER et coll., KÄUFER et coll. sowie TROUPP & HEIS

KANEN auch auf einen Zirkulationsstillstand in den Vertebralarterien hinwiesen. Die meisten Untersuchungen erfolgten aus diagnostischen Gründen und seltener unter dem Aspekt der zerebralen Todeszeitbestimmung. KAUFER et coll. wiesen intrazerebrale Zirkulationsstillstände nach Hypoxydosen, also bei Hirntod infolge extrazerebraler Noxen, nach.

Da wir unter dem Hirntod den irreversiblen Funktionsuntergang des gesamten Gehirns, also von Hirnrinde und Stammhirn, verstehen (PENK & KÄLFER), muß zur Sicherung der Diagnose eine Darstellung der Versorgungsgebiete beider Karotiden und der Arteria basilaris erfolgen. Die alleinige Durchführung der Karotisangiographie, die bislang zur Todeszeitbestimmung des Zerebrums gefordert wurde, reicht nach unserer Meinung zur Klärung dieser Frage nicht aus. Wegen der Versorgung des Stammhirns über die Vertebralarterien muß ferner in den Fällen, bei denen ein extrakranieller Kontrastmittelstopp in einer Vertebralarterie vorliegt, immer die kontralaterale Arterie dargestellt werden, um mit Sicherheit einen Zirkulationsstillstand im Stammhirngebiet zu beweisen. Dagegen kann man bei einer Kreislaufunterbrechung in der Arteria basilaris auf die Angiographie der zweiten Vertebralarterie verzichten. Ferner ist darauf zu achten, daß unter Berücksichtigung eines Sicherheitsintervalles von 30 Minuten eine erneute Karotis- und Vertebralsangiographie durchgeführt wird, wenn sich nicht die Gesamtdauer der Untersuchung über diesen Zeitraum erstreckt. Damit soll die Diagnose des Hirntodes durch einen zerebralen Zirkulationsstillstand jenseits der Grenze, bei der eine Restitution der Hirnfunktionen mit Sicherheit unmöglich geworden ist, exakt abgesichert werden.

Die Erfüllung der genannten Forderungen, die Voraussetzung einwandfreien Hirntodbestimmung sind, wird nach unseren Erfahrungen mit den verschiedenen radiologischen Untersuchungsmethoden, die miteinander verglichen wurden, am besten durch die transfemorale selektive Katheterangiographie der zuführenden Hirnarterien gewährleistet.

Wenn auch keine wesentlichen diagnostischen Unterschiede zwischen den durch Direktpunktion und Katheteruntersuchung gewonnenen Angiogrammen besteht, so erscheint uns die Katheterangiographie aus folgenden Gründen vorteilhafter: (1) Durch Verwendung eines Spezialkatheters können in einem Untersuchungsgang beide Karotiden und Vertebralarterien selektiv nacheinander dargestellt werden. Dadurch wird der zeitliche Aufwand gegenüber den Direktpunktionen deutlich vermindert, vor allem entfallen die schwierige Direktpunktion der Arteria vertebralis oder eine zusätzliche Untersuchung, z. B. eine Gegenstrominjektion. Nur in wenigen Fällen mißlingt die Sondierung aller 4 Arterien mit einem Katheter. In diesen Situationen müssen dann ein Katheter wechseln oder eine Direktpunktion durchgeführt werden. (2) Der Katheter



Abb 1 Schußverletzung des Schädels a) Karotisangiogramm rechts. Abbruch der Kontrastmittelsäule an der Schädelbasis bei normaler Darstellung der Arteria carotis externa und ihrer Äste b) Vertebroangiogramm rechts. Kontrastmittelstopp in der Arteria basilaris. Retrograde Füllung der gegenseitigen Arteria vertebralis c) Vortorsozervikale Angiographie (10 Minuten nach b). Kontrastmittelstopp im zervikalen Bereich der Arteriae carotides internae und vertebrales

kann ohne Komplikationsgefahr über einen längeren Zeitraum im Gefäß liegen bleiben. Die Bildung von Thromben wird durch häufiges Durchspülen des Katheters verhindert (3). Vorteilhaft erscheint ferner, daß nach der zerebralen Angiographie mit dem selben Katheter eine Nieren- oder Leberangiographie zur Klärung der Gefäßtopographie für eine prospektive Transplantation durchgeführt werden kann.

Letztlich hängt der Untersuchungsmodus von der gefäßanatomischen Gesamtsituation der Arterien ab. d. h. bei Stenosen oder Verschlüssen wird man auf eine Direktpunktion zurückgreifen müssen.

Eine Alternative zu den Katheterv Verfahren und den Direktpunktionen bedeuten die einfacher und schneller durchführbare Panangiographie des Gehirns mit Injektion einer größeren Kontrastmittelmenge in die Aorta ascendens oder die Gegenstrominjektion von der Arteria brachialis. Vergleichende Untersuchungen mit der Katheterangiographie und Panangiographie haben jedoch gezeigt, daß die auf Katheterangiogrammen einwandfrei nachweisbaren Kontrastmittelunterbrechungen bei der Panangiographie aufgrund des schwächeren Kontrastes nicht immer exakt erkennbar waren. Das gilt vor allem für den intrazerebralen Zirkulationsstopp. Ferner war die Kreislaufunterbrechung bei

der Panangiographie immer tiefer als bei den selektiven Methoden lokalisiert. Diese Tatsache deutet darauf hin, daß bei den selektiven Methoden durch die erhöhte Druckinjektion das Kontrastmittel weiter nach kranial gepreßt wird. Nach unserer Meinung wird in absehbarer Zeit, wenn weitere Erfahrungen durch vergleichende Untersuchungen gewonnen worden sind, zur Bestimmung des Hirntodes die aortozervikale Angiographie ausreichen.

### Ergebnisse

Unsere Befunde basieren auf 43 Hirntodeszeitbestimmungen. In 23 Fällen wurden mit der Katheterangiographie alle zuführenden Hirnarterien dargestellt und 11 Wiederholungsuntersuchungen durchgeführt. Alle Katheteruntersuchungen erfolgten unter standardisierten Bedingungen und außerdem zum Teil aus Vergleichsgründen mit manueller und maschineller Kontrastmittelinjektion. Wesentliche Unterschiede in der Lokalisation des Stopps konnten dabei nicht festgestellt werden. Bei 8 Patienten kam außerdem eine zerebrale Panangiographie zur Anwendung. In den übrigen Fällen erfolgten entweder Direktpunktionen der Karotiden und elektive Injektionen in die Vertebralarterien bzw. die Arteria subclavia nach transfemoraler oder transaxillärer Katheter-einführung.

Bei der zerebralen Serienangiographie ergaben sich folgende Befunde: (1) Stromungsverlangsamung in der Arteria carotis interna und der Arteria basilaris, wobei die intrazerebralen Äste Lumenvershmälerungen aufweisen konnten, sowie Fehlen einer venösen Phase im Karotis- und Vertebralsangiogramm. (2) Spitz zulaufender Abbruch der Kontrastmittelsäule in den Anfangsabchnitten der Arteria cerebri media sowie in den Ästen der Arteria basilaris (Abb. 3 a, 4 b) ohne Kontrastierung der peripheren Abschnitte. (3) Kontrastmittelstopp im Karotissiphon (Abb. 1 a, 2 a) oder an der Schädelbasis (Abb. 1 a) sowie in der kaliberreduzierten Arteria basilaris (Abb. 1 b) oder an der Schädelbasis mit gelegentlicher Darstellung der kontralateralen Arteria vertebralis. (4) Zirkulationsunterbrechung im zervikalen Abschnitt der Arteria carotis interna (Abb. 3 b) und in der Arteria vertebralis am atlanto-oxzipitalen Übergang oder im Halsbereich.

In allen genannten Punkten läßt sich eine normale Füllung der Arteria carotis interna und ihrer Äste (Abb. 1 a, 2 a, 3 b, 4 a) sowie der extrakraniellen Äste der Vertebralarterien (Abb. 1 b, 2 b, 3 a, 4 b) nachweisen, die im Vergleich zu der verzögerten Kontrastierung der intrakraniellen Gefäßregionen der Arteria carotis interna und der Arteria basilaris besonders auffallend ist. Ebenso wie die Befunde weisen die Diskrepanz zwischen den lumenreduzierten intrakraniellen und den normalkalibrigen extrazerebralen Arterienregionen



Abb 2 (Legende auf der gegenüberstehenden Seite)



Abb 3 (Legende auf der gegenüberstehenden Seite)



Abb 4 Schädelhirntrauma Zustand nach Trepanation a) Karotisangiogramm rechts Kontrastmittelstopp im zervikalen Abschnitt der Arteria carotis interna bei normaler Füllung der Arteria carotis externa. b) Vertebralsangiogramm links (10 Minuten nach a) Abbruch der Kontrastmittelsäule in den Ästen der kalibrierten Arteria basilaris Darstellung des Karotissiphons der Anfangsabschnitte der Arteria cerebri media und der Arteria ophthalmica über den Ramus communicans posterior

owie eine lange Stase des Kontrastmittels vor dem Stopp auf eine Erhöhung des zerebrovaskulären Widerstandes hin. Vor allem erscheint die protrahierte Verweildauer des Kontrastmittels in den kleineren Hirnarterien, an denen besonders bemerkenswert wobei wir den Kontrast über Zeiträume bis zu 30 Minuten bei nachfolgenden Untersuchungen beobachten konnten (Abb 3).

Die Zirkulationsunterbrechungen in der Arteria carotis interna im Siphonbereich an der Schädelbasis sowie der Stopp in der Arteria basilaris bzw.

Abb 2 Hypoxydase a) Zirkulationsunterbrechung im Karotissiphon bei normaler Externafüllung Darstellung der Arteria ophthalmica. b) Vertebralsangiogramm (10 Minuten nach a) Darstellung der Arteria basilaris und ihrer Äste

Abb 3 Schädelhirntrauma. a) Vertebralsangiogramm rechts Spitz zulaufender Kontrastmittelaufbruch in den Ästen der Arteria basilaris Lumenverschmälerung der Arteria basilaris Kontrastierung des Ramus communicans posterior b) Karotisangiogramm rechts (10 Minuten nach a) Abbruch der Kontrastmittelsäule im zervikalen Abschnitt der Arteria carotis interna bei normaler Füllung der Arteria carotis externa. Restfüllung der Arteria basilaris und ihrer Äste des Ramus communicans posterior und der Arteria cerebri media von der vorausgegangenen Vertebralsangiographie



vertebralis sind beweisende und zuverlässige Kriterien für den Hirntod. Eine Kontrastierung der Arteria ophthalmica, die wir in der Hälfte der Fälle feststellen konnten (Abb. 2 a), spricht nicht gegen eine intrakranielle Zirkulationsunterbrechung, da die Stromungsverhältnisse zwischen den intrazerebralen Karotisabschnitten und der Arteria ophthalmica nicht korrelieren (TONNIS & FROWEIN). Gleichartig zu bewerten sind Zirkulationsstillstände im zervikalen Abschnitt der Arteria carotis interna und der Arteria vertebralis, die durch eine Stagnation der Blutsäule vor dem funktionell intrazerebral lokalisierten Verschluss erklärt werden können. Allerdings müssen in dieser Lokalisation die angiographischen Befunde insofern vorsichtiger interpretiert werden, als hier vor allem bei der Arteria carotis interna Prädispositionsstellen für organische Gefäßverschlüsse liegen.

Bei einer rudimentären Darstellung der Anfangsabschnitte der Arteria cerebri media und der Arteria cerebri posterior sowie bei einer Zirkulationsverlangsamung über 14 Sekunden mit Fehlen der venösen Phase im Karotis- und Vertebralisangiogramm erscheint die Deutung der Befunde im Sinne des Hirntodes kritischer, da hierbei das Faktum der interzerebralen Kreislaufunterbrechung nicht erfüllt ist. Nachdem im weiteren Verlauf einer Hirnschädigung das Hirnödem ständig zunimmt und damit der zerebrovaskuläre Widerstand progredient ansteigt, darf man für diese Situationen unterstellen, daß die Angiographie zu früh erfolgte. Der spitzzipflige glatte Gefäßabbruch in den kleinen intrazerebralen Arterien und die lange Stase des Kontrastmittels deuten darauf hin, daß dieses Röntgenphänomen Folge des unphysiologischen erhöhten Injektionsdruckes ist. Diese druckmechanische Vorstellung wird dadurch untermauert, als bei der vergleichsweise durchgeführten Panangiographie (Abb. 1 c) keine Darstellung dieser Regionen erzielt werden konnte. Selbst wenn man die rudimentäre Kontrastierung der kleinen intrazerebralen Arterien noch als axiale Minimalstromung auffassen würde, dürfte mit dieser extremen Zirkulationsverlangsamung keine funktionell ausreichende Hirndurchblutung vorliegen, da sich die Stromungsverlangsamung weniger stark im arteriellen als im kapillaren und venösen Bereich der Hirnzirkulation auswirkt.

Durch unsere zum gleichen Zeitpunkt durchgeführten Karotis- und Vertebralisangiographien läßt sich zwischen dem Zirkulationsstillstand in den Arteriae carotides und in den Arteriae vertebrales bzw. in der Arteria basilaris oft ein unterschiedlich langes zeitliches Intervall feststellen (Abb. 2 3 4). Wir konnten bei einem Kontrastmittelstopp in beiden Karotiden im Bereich des Siphons an der Schädelbasis oder im zervikalen Abschnitt noch eine Durchströmung der Arteria basilaris und eine rudimentäre Füllung ihrer Äste nachweisen. In 3 Fällen kam es sogar zur Darstellung der Arteria carotis interna über den Ramus communicans posterior bei einem Karotisstopp im zervikalen Gefäßab-

schnitt (Abb 3, 4) Die zeitlich später einsetzende Zirkulationsunterbrechung im Vertebralis und Basilariskreislauf beinhaltet daß nach dem Untergang des Großhirns der Verlust der Stammhirnfunktionen zu einem späteren Zeitpunkt einsetzt Da wir unter Hirntod den irreversiblen Funktionsverlust des gesamten Gehirns verstehen kann dieser erst nach Feststellung eines Kontrastmittelstopp in der Arteria basilaris oder in den Vertebralarterien definitiv angenommen werden

Das entscheidende Problem für die endgültige Feststellung des zerebralen Todes besteht daher in der zeitlichen Koordinierung zwischen Nulllinien EEG und dem optimalen Zeitpunkt für die Angiographie Wird die Gefäßdarstellung unmittelbar nach Feststellung eines Nulllinien EEG durchgeführt, so läßt sich in die frühen Stadium der Entwicklung noch eine intrakranielle Durchströmung nachweisen Sicherlich erlebt man hier variable Situationen für die je nach der Ausdehnung eines Hirnschadens ein lokales oder generalisiertes Hirnodem mit unterschiedlich rascher Ausbildung verantwortlich ist Jedenfalls werden dadurch Wiederholungsangiographien erforderlich um durch die Kreislaufunterbrechung in sämtlichen Hirnarterien an der Schadelbasis oder in den zervikalen Regionen den Hirntod zweifelsfrei zu beweisen

Zur exakten Terminierung des optimalen Untersuchungszeitpunktes für die Angiographie erscheint uns die AVDO wertvoll Beim Funktionsuntergang des Zerebrums kommt es zur starken Abnahme der AVDO die auf einen fehlenden Sauerstoffverbrauch des Gehirns schließen läßt Nach tierexperimentellen Untersuchungen (HIRSCH et coll.) tritt jedoch keine absolut gehobene AVDO auf da ein geringer Sauerstoffverbrauch durch überlebende Gliazellen und Anastomosen aus dem Karotis-externa-Kreislauf bestehen bleibt Wegen des Kreislaufstillstandes beim Hirntod lassen sich zudem keine exakten Werte der AVDO gewinnen so daß die Methode keine weiteren Aussagen über den zerebralen Funktionszustand erlaubt Der Wert dieser Untersuchung liegt unseres Ermessens darin daß der im Verlauf nach anfänglichem Abfall nachweisbare Wiederanstieg der AVDO durch retrograde Blutaspiration von peripherem Blut in die Vena jugularis als Hinweis auf einen eingetretenen Zirkulationsstillstand dienen kann und somit als Indikator für den optimalen Zeitpunkt der Angiographie zu werten ist

## ZUSAMMENFASSUNG

Klinisches Syndrom Nulllinien EEG und erniedrigte AVDO<sub>2</sub> bilden die Voraussetzung zur Durchführung einer zerebralen Angiographie die zur Zeit allein mit der genügenden Sicherheit die Diagnose des zerebralen Todes erlaubt. Darunter verstehen wir den irreversiblen Funktionsuntergang des Gesamthirns so daß diese Definition eine Angiographie beider Karotid- und Vertebralarterien erfordert Nach unseren Erfahrungen an 43

Patienten betrachten wir als Kriterium des Hirntodes im Karoti Angiogramm einen Kontrastmittelstopp in Höhe des Siphons an der Schädelbasis oder im zervikalen Abschnitt der Arteria carotis interna bei normaler Füllung der Arteria carotis externa und im Vertebralisangiogramm einen Zirkulationsstillstand in der Arteria basilaris bzw. vertebralis. Damit der zervikalen Serienangiographie im zeitlichen Intervall nachweisbaren Zirkulationsunterbrechungen sind beweisend für den irreversiblen Funktionsuntergang des gesamten Gehirns, denn nach dieser länger dauernden Anoxie des Zerebrums ist keine Restitution zu erwarten. Die Zirkulation im Versorgungsgebiet der Arteria basilaris sistiert zu einem späten Zeitpunkt als die im Bereich der Arteria carotis interna.

## SUMMARY

Clinical findings a zero line ETG and a reduced pO<sub>2</sub> A/D are indications for cerebral angiography which alone proves cerebral death. This signifies irreversible loss of cerebral function. Bilateral vertebral and carotid angiography in 43 cases revealed this loss by demonstrating arrest of the contrast medium at the level of the siphon at the base of the skull or in the cervical part of the internal carotid artery with normal filling of the external carotid artery and obstruction in the basilar artery or the vertebral artery itself. The circulatory arrests demonstrable are proof of irreversible cerebral death as the ensuing cerebral anoxia eliminates any possibility of recovery of cerebral function. The vertebral circulation ceases later than the carotid blood flow.

## RÉSUMÉ

Le syndrome clinique ETG plat et l'abaissement de la différence arterioveineuse de pression d'oxygène constituent les préfabriques à l'exécution d'une angiographie cérébrale qui actuellement permet seule avec une sécurité suffisante le diagnostic de mort cérébrale. Nous entendons par là la défaillance fonctionnelle irréversible de l'encéphale entier, de sorte que cette définition nécessite une angiographie des deux artères carotides et des deux artères vertébrales. D'après notre expérience sur 43 patients nous considérons comme critères de la mort cérébrale sur l'angiographie carotidienne un arrêt du moyen de contraste au niveau du siphon à la base du crâne ou dans la partie cervicale de l'artère carotide interne alors qu'il y a un remplissage normal de l'artère carotide externe et sur l'angiographie vertébrale un arrêt circulatoire dans l'artère basilaire ainsi que dans la vertébrale. Les interruptions de circulations qui sont constatées pendant la durée de l'angiographie cérébrale en série sont la preuve de la défaillance fonctionnelle irréversible de l'ensemble du cerveau car après cette anoxie cérébrale d'assez longue durée il n'y a plus à espérer aucune restitution. La circulation dans le territoire de l'artère basilaire s'arrête plus tardivement que dans le territoire de l'artère carotide interne.

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Abb. 2 Fall 2 a) Caroti angiogramm rechts Die A. carotis füllt sich nur bis zur Schadelbasis retrograde Füllung der infratentoriellen Gefäße b) Angiogramm nach Infusion von Mannit Darstellung normaler intracraneller Gefäße

**Fall 2** 18 Jahre männlich Seit Oktober 1968 morgendlich Grand mal Anfälle später häufig Absencen Im November erstmals stationäre Behandlung Neurologisch kein krankhafter Befund im EEG krampfverdächtige Potentiale ohne Herdhinweis Therapie mit Mylep in etwa 1 Jahr später erneute Anfallsreien mit Somnolenz Bei der Aufnahme auch diesmal neurologisch unauffällig Bei weiterer Verschlechterung der Bewußteinslage seitengleichem Reflexverhalten beiderseits positiven Pyramidenbahnzeichen wurde zur weiteren Abklärung eine Angiographie durchgeführt Dabei wurden intracerebrale Gefäße nur bis zur Teilungsstelle der A. carotis interna dargestellt Gleichzeitig füllten sich die infratentoriellen Gefäße Nach Infusion von Mannit kam es bei der Kontrollangiographie zu einer guten Füllung aller Carotisäste Durch weitere entwässernde Therapie und Anticonvulsiva besserte sich der Zustand des Patienten bald so daß er nach Hause entlassen werden konnte

Nach einigen Monaten erneute Aufnahme im Status epilepticus Während des klinischen Aufenthaltes verschlechterte sich der Bewußteinszustand zunehmend und der Patient verstarb nach einigen Tagen Bewußtlosigkeit an rezidivierenden Pneumonien Bei der Sektion waren beide Carotiden frei es fand sich ein Hydrocephalus internus et externus (Herrn Prof. W. Müller Direktor des Pathologischen Instituts Essen danken wir für die überlassenen Sektionsbefunde)

**Fall 3** 27 Jahre weiblich Bisher nie ernsthaft krank gewesen Seit Januar 1970 nach einer Crippe Kopfschmerzen seit Mitte Mai flüchtige wechselseitige Paresen zunehmende Vergeßlichkeit Einweisung in die Klinik in somnolenter Bewußteinslage Die adipose

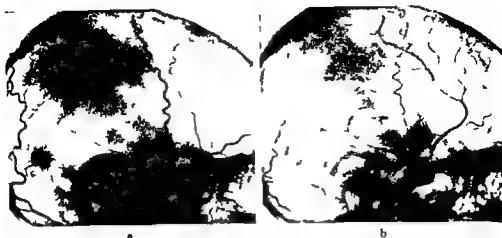


Abb 3 Fall 3 a) Carotisangiogramm rechts Keine Darstellung intracranießer Gefäße gute Füllung des Externakreislaufs b) Angiogramm nach Infusion von Sorbit. Darstellung der Hirngefäße Verschluss der A. cerebri media

Patientin reagierte bei der Aufnahme nur auf Schmerzreize Pupille links weiter als rechts beiderseits nur träge Lichtreaktion Es bestand eine beiderseitige Muskeltonuserhöhung mit Streckkrämpfen Muskelsehnenreflexe seitengleich Babinski beiderseits positiv Blutdruck 120/80 mm Hg Puls 80/min Die Atmung war regelmäßig Bei der Untersuchung erbrach die Patientin Im weiteren Verlauf zunehmende Nackensteifigkeit Lumbalpunktion 64/3 Zellen der Liquor war klar Das Echo war mittelständig

Bei der Carotisangiographie kam es zu keiner intracranialen Gefäßfüllung erst nach Infusion von Sorbit ließ sich eine gute Gefäßdarstellung erreichen Die A. pericallosa war mittelständig es bestand ein Verschluss der A. cerebri media rechts

Der Allgemeinzustand der Patientin verschlechterte sich zunehmend am folgenden Tage kam sie im zentralen Atemstillstand ad exitum Bei der Sektion waren beide Carotiden frei es fanden sich Zeichen eines erhöhten Hirndrucks Der Mediaverschluss rechts wurde bestätigt

### Diskussion

Unsere Beobachtungen decken sich bezüglich Ätiologie Symptomatik und Prognose mit den in der Literatur angegebenen Fällen Wenn man dabei auch meistens ein isoelektrisches EEG findet so wurde doch wiederholt beobachtet (KOLER 1969 LORENZ 1969) daß dieser Befund einen Hirntod nicht beweisen kann sondern nur Ausdruck einer schweren cerebralen Funktionsstörung ist Erst eine EEG Kontrolle nach Ablauf von 12 Stunden mit gleichem Befund bestätigt eine irreversible Schädigung

Der Nachweis eines vollständigen intracranialen Kreislaufstop wäre ein sicheres Kriterium für den Hirntod doch ist dieser nicht ohne weiteres zu

führen. Wenn es auch bei der Angiographie nicht zur Darstellung der Hirngefäße kommt, so ist damit eine Stase noch nicht bewiesen. Wie wir bei 3 Patienten nach intravenöser Gabe von Sorbit zeigen konnten, kam es kurze Zeit nach der Entwässerung bei erneuter Angiographie zu einer Füllung der Hirngefäße. Obwohl bei der primären Untersuchung das Kontrastmittel nur bis zur Schädelbasis nachweisbar war, mußte doch noch eine — wenn auch geringe — intracranelle Blutzirkulation stattgefunden haben, denn sonst wäre eine entzündende Wirkung des Sorbit im Hirn nicht möglich gewesen. Ein Dauererfolg ließ sich mit dieser Therapie nicht erzielen. Nach kurzer Zeit traten Atemstillstand oder Kreislaufchock ein. Eine ähnliche Beobachtung machten FROWEIN *et coll.* (1970) bei einem Patienten, der mit Harnstoff behandelt wurde.

In welchem Umfang die Angiographie der Hirngefäße als Todesnachweis herangezogen werden kann, läßt sich aufgrund unserer eigenen Erfahrungen noch nicht sagen. Doch wir glauben mit ADAMS *et coll.* (1969), BROCK *et coll.* (1969) und anderen Autoren (GROS *et coll.* 1969, KUELFER 1969, LORFAZ 1969, WULFENWEBER 1969) darin übereinzustimmen, daß die Angiographie neben anderen Methoden angewandt werden sollte, daß sie jedoch allein kein zuverlässiger Nachweis des cerebralen Todes ist. Einige Autoren (BROCK *et coll.* 1969, GOODMAN 1969) fordern die dauernde Abwesenheit einer Clearance eines in das Gehirn eingebrachten radioaktiven Isotops als sicheren Nachweis einer fehlenden intracranellen Blutzirkulation, um damit den definitiven Hirntod festzustellen.

## ZUSAMMENFASSUNG

Wenn es auch bei einer Angiographie nicht zu einer Darstellung der Hirngefäße kommt, ist ein vollständiger Kreislaufstop noch nicht bewiesen. Bei einigen Patienten kam es kurze Zeit nach intravenöser Infusion von Sorbit bei erneuter Angiographie zu einer Füllung der Hirngefäße. Es mußte also noch eine angiographisch nicht nachweisbare Blutzirkulation stattgefunden haben. Aus diesem Grund halten wir eine fehlende Gefäßdarstellung der intracranellen Gefäße nicht beweisend für den Eintritt des Hirntodes. Die Angiographie kann zur Unterstützung anderer Methoden hinzugezogen werden.

## SUMMARY

Proof of a complete arrest in the circulation is not necessarily obtained by the failure of angiography to demonstrate the cerebral vessels. These may sometimes be filled by repeat angiography following the intravenous infusion of Sorbit. It would therefore appear that some degree of circulation then still exists though it may not be ordinarily demonstrable. Such failure of angiography cannot therefore be regarded as evidence of cerebral death. The role of angiography is only a supporting one applicable in conjunction with other methods.

## RÉSUMÉ

L'absence de visibilité des vaisseaux cérébraux au cours de l'angiographie n'est pas une preuve absolue d'arrêt de la circulation. Chez certains patients une nouvelle angiographie faite peu de temps après une injection intraveineuse de Sorbitol a donné un remplissage des vaisseaux cérébraux. Il y avait donc nécessairement une circulation sanguine même si elle ne pouvait pas être mise en évidence par l'angiographie. C'est pourquoi nous considérons que le défaut de visualisation des vaisseaux intracrâniens n'est pas la preuve de l'installation de la mort cérébrale. L'angiographie peut être utilisée pour confirmer les résultats d'autres méthodes.

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## CRITERIA OF CEREBRAL DEATH

by

Y. MIYAZAKI, H. TAKAMATSU, Y. IANAKA, N. MIKAMI,  
S. ARAKAWA and I. SOHMA

The recent advent of organ transplantation including that of the heart, has moved the emphasis of the evidence of death of the donor from cardiac arrest to cessation of cerebral function. The decision as to whether changes in brain function are reversible or irreversible is not only necessary from the view point of donor selection, but is also required for deciding the limit of artificial respiration in the field of neurosurgery.

The authors have had a considerable number of neurosurgical cases in which ordinary breathing ceased and which required artificial aid. Forty-two cases over the past three years that required artificial respiration for more than 24 hours were investigated. As a result the significance of an abrupt fall in blood pressure as one of the indications of cerebral death became apparent. The authors therefore decided to conduct experimental investigations in dogs. It appeared that a sudden drop in pressure resulted from paralysis of the vasomotor centre in the brain stem. It is now stressed that this sign should be included as one of the important criteria for the decision of cerebral death. A method of deciding death of the brain that has been developed by the authors will also be reported.

The examination of brain stem function is important as an indication of cerebral death. SCHWAB et coll (1963), HAMILIN (1964), HOCKADAY et coll (1964), STANN et coll (1967) and ROSOFF & SCHWAB (1968) emphasized the significance of an electroencephalogram (EEG). HOCKADAY et coll made a classification of EEG

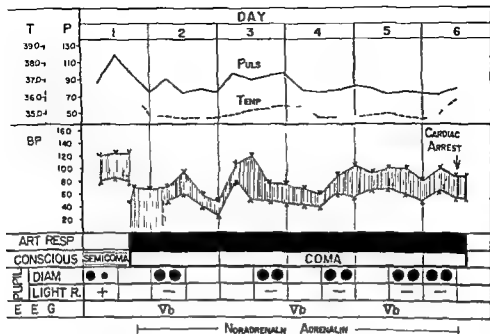


Fig 1 A case of severe head injury

and reported that his grade Va or Vb may be utilized in deciding upon death of the brain. Although recovery may be expected with an isoelectric EEG as described by several authors (FOIS et coll 1955, FISCHGOLD & MATHIS 1957, PEARCY & VIRTUE 1959, CAVAFUTTI 1959, BENTA & LEIBOWITS 1961, KUBICKI 1966, LEVY & KINVEL 1966) there is some doubt as to whether this is of absolute value in the conclusion. A combination of other signs should therefore be considered; a finding based upon the EEG alone would be dangerous.

The authors have been advocating that cerebral death should mean irreversible loss of function of the whole brain including the brain stem. The function of the latter, which is directly connected with the maintenance of life, is associated with respiration and the vascular circulation, the former governed by the respiratory centre and the latter by the vasomotor centre. Cases encountered in the neurosurgical field usually have cessation of ordinary breathing and require artificial respiration. It is evident in these that the function of the respiratory centre of the brain stem has been lost and it is therefore important to determine whether the function of the vasomotor centre in the brain stem persists. The vasomotor centre controls the heart and almost the entire body arterial system, the arterial tone is maintained and the blood pressure kept at a constant level.

A disturbance in function of the vasomotor centre therefore produces a rapid fall in the latter.

The cessation of ordinary respiration arises from an intracranial process producing direct or indirect damage of brain stem function. It is thus an important target in the determination of cerebral death.

Forty-two recent cases of cessation of ordinary breathing, that required artificial respiration exceeding 24 hours were considered to determine whether an abrupt fall in blood pressure had occurred and to determine its time of onset. The 42 cases were divided into six groups. Group 1: Cessation of ordinary breathing simultaneously with fall in blood pressure. Group 2: Cessation of ordinary breathing and fall in blood pressure within one hour. Group 3: Cessation of ordinary breathing, and fall in blood pressure after one hour. Group 4: Fall in blood pressure and cessation of ordinary breathing. Group 5: Cessation of ordinary breathing alone. Group 6: Miscellaneous.

The first group (Fig. 1) consisted of cases in which a sudden fall occurred almost at the same time as the cessation of ordinary respiration while in the second and third groups the drop was recorded within one hour and over one hour respectively later. In contrast to the second and third groups, the fourth group in spite of no cause such as blood loss first had a steep fall in pressure after which respiration ceased. The fifth group consisted of cases in which cessation of ordinary breathing alone was observed without any change in blood pressure for several days. The sixth group was made up of cases referred from other hospitals which at the time of admission had already passed through the point of an abrupt drop in pressure the time of which was unknown. The sixth group also included the cases in which a sudden fall occurred but in which the pressure recovered and was normally maintained without the administration of vasopressor agents. The cases in which a steep drop was evident after the cessation of ordinary breathing belong to the first, second and third groups and reach a total of 29 cases (69 per cent of the entire number) (Table).

While a sudden fall in blood pressure during intensive care of the patient appeared to be no more than moderate it cannot, because of the danger be neglected. On the other hand the lives of only 2 of 4 cases of group 5 in which a drop failed to be recognized were saved. The remaining 10 cases developed cardiac arrest while there were some differences in the length of the course. From the facts it is in the field of neurosurgery in which the cessation of ordinary breathing alone occurs that a possibility of saving the patient by radical or symptomatic treatment by artificial respiration exists. However when the cessation of spontaneous respiration is accompanied by a steep fall in blood pressure, the latter appears later or the pressure can be maintained only by vasopressor agents the prognosis is extremely poor.

Table  
*Frequency and prognosis in the six groups*

Group	No of cases	Per cent	Prognosis	
			Alive	Dead
I	15	35.7	0	15
2	4	9.5	0	4
3	10	23.8	0	10
4	2	4.8	0	2
5	4	9.5	2	2
III	7	16.7	0	7
Total	42	100.0	2	40

The ECG recordings at the time of an abrupt lowering of blood pressure appeared to be normal. This suggested that the fall cannot be attributed to disturbance of cardiac function such as the lowering of output. A possibility however exists that acute cardiac failures may not produce immediate changes in the ECG. It therefore becomes necessary to prove that the cause lies in the failure of the vasomotor centre in the brain stem by establishing that a sudden decrease in pressure arises from the lowering of the tone of the walls of the peripheral arteries.

The authors when confronted with such a fall in blood pressure administered various vasopressor agents by different mechanisms in an attempt to raise and maintain it. Proternol which stimulates the  $\beta$  system produced no effect while adrenalin and noradrenalin which affect the walls of the peripheral arteries and causes them to contract were satisfactory. The clinical experiences appear to confirm that the fall may be caused by the lowering of resistance to the peripheral blood flow coming in turn from the decrease in tone of the peripheral arterial wall.

An experimental investigation was carried out to determine whether brain stem damage in animals would produce a sudden drop in blood pressure in addition to an arrest of breathing as occurs clinically and whether such a fall arose from diminished cardiac action in brain stem damage.

A transclival approach to the ventral surface of the pons and medulla oblongata was made in 130 adult mongrel dogs. MONVIER (1939) and ALEXANDER (1946) in their electric stimulation experiment determined the location of the vasomotor centre in the cranial and caudal aspect of the obex. The present authors therefore elected a site 3 mm cranial to the obex the junction of both



Fig. 3. Lesion in the brain stem of a dog.

the anterior branches of the cerebro-pinal artery and the basilar artery together with the brain stem tissue were included in the centre of damage (Fig. 2). This damage to the brain stem tissue as in Fig. 3 necessitated immediate artificial respiration following which ordinary breathing failed to reappear. On the other hand, the blood pressure first rose then gradually fell with the lapse of time; the pressure usually returned in four to five minutes to its value before the brain stem damage, to fall again until it reached 50 to 40 mm. Measurements continued in the follow-up period failed to record values lower than 50 to 40 mm. This 40 mm pressure probably represents that of the heart and is maintained by peripheral control of the tone of the arterial walls.

Damage to the brain stem at the site corresponding to the vasomotor centre described by MONVIEFF and ALEXANDER together with the irreversible cessation of ordinary breathing produced a tentative rise in blood pressure followed by a fall to 50 to 40 mm.

The outstanding blood pressure falls as experienced in the authors' clinical cases occurred also in brain stem damage experiments. Moreover the fact that their appearance followed cessation of ordinary breathing seems to suggest that the falls in clinical cases arise from loss of the brain stem function.

Continuous and sustained recording of the blood pressure before brain stem damage and by electric stimulation of the cranial nerve produces a definite rise in blood pressure. Repeated electric stimulation of the cranial nerve in the course of the lowering of blood pressure in brain stem damage failed however to raise or inhibit the fall (Fig. 4). This indicates that the rise reflex of blood pressure by peripheral nerve stimulation was lost and substantiates the suggestion that brain stem reaction was absent.

Fig. 5 presents the results obtained by the administration of 0.5 mg. noradrenalin in lowered blood pressure produced by brain stem damage. The definite blood pressure rise confirms the findings in clinical cases.

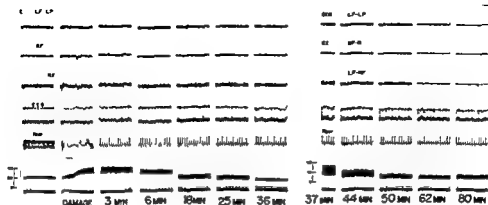


Fig 3 Recording of EEG ECG respiration and blood pressure before and after damage to the brain stem (vasomotor centre)

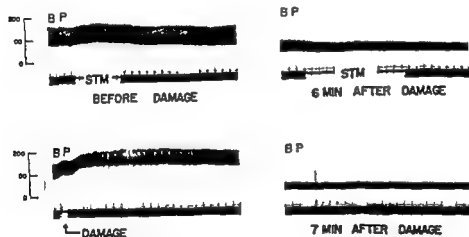


Fig 4 Effect of sciatic nerve stimulation on blood pressure

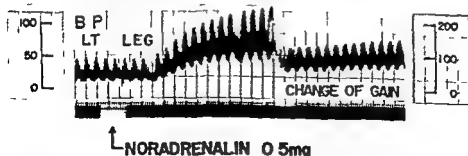


Fig 5 Effect of noradrenalin on lowered blood pressure arising from brain stem damage

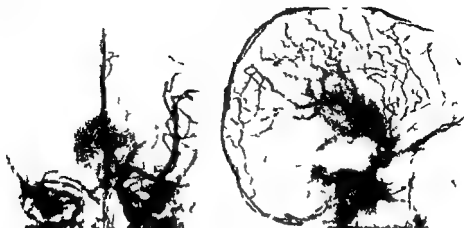


Fig. 11. Case of intraventricular tumour. First cerebral angiogram.

An attempt was made to clarify that the blood pressure drop from brain stem damage is not influenced by cardiac output but rather comes from the lowering in tone of the peripheral arterial walls. A femoral artery of a brain stem damaged dog was cross-circulated by the arterial blood of a healthy dog (pump dog). The nerve distribution of the femoral arterial wall of the dog to be subjected to brain stem damage was thus left intact and the arterial blood flow alone circulated by the blood flow of the pump dog. No other operation loads were thus inflicted upon the pump dog except for the insertion of polythene tubes into the artery and vein. The cardiac output failed to change proving that the blood pressure of the unilateral femoral artery of the experimental dog under cross-circulation was subject only to the tone of the arterial wall. The femoral arterial pressure of the blood under cross-circulation in the pump dog was lowered by brain stem damage and indicated that the fall arose from lowering in tone of the arterial wall. In other words the cause was the loss of function of the vasomotor centre.

Carotid and vertebral angiographies were performed in cases considered to be of cerebral death as well as before and after the critical point of such death. Non-filling of the intracranial arteries was observed in the second and third angiographies (Figs 6-7). It would therefore appear that cerebral angiography should be repeatedly carried out to prove non-filling of the cerebral arteries. As regards the degree of non-filling of the internal carotid artery it was noted that the part distal to the ophthalmic artery was not filled but that the ophthalmic artery itself was sometimes outlined. In other cases the cavernous part of the internal carotid artery was not filled with the ophthalmic artery appearing the same. There was thus no uniformity.



Fig 7 Same case as in fig 6 Second cerebral angiogram with non filling of the intracranial arteries

Every effort has been made not to overemphasize certain points in the authors criteria of cerebral death

The kind of original disease and its course the clinical neurologic findings EEG findings and changes in brain stem function are collectively judged to determine whether the patient has entered the state of cerebral death The determination as to whether a patient is in this state or not gives rise to the problem of when the examinations should be started Patients examined for cerebral death are in a coma and receiving artificial respiration As for the time to initiate positive examinations such as EEG recordings and the establishment of the presence or absence of central nervous system function this seems to be about 24 hours after continued artificial respiration The time point 24 hours after the initiation of artificial respiration was decided from the authors clinical experiences and with due respect to the psychologic factors of the patient's family This time appears to be particularly important in a case of sudden onset of coma and respiratory arrest

When the original condition follows a comparatively long course and is aggravated and especially when the cessation of normal breathing can be predicted it may not be absolutely necessary to adhere to 24 hours and permissible to initiate the enquiry into cerebral death earlier



The nature of the original condition leading to coma and arrest of breathing is highly important in any decision regarding cerebral death. The original conditions may be divided into central nervous system disease (e.g. severe brain injury, brain tumour, cerebral vascular disease) and diseases primarily of the heart (e.g. severe cardiac disease) and temporary cardiac arrest arising from various causes during operation. The prognosis regarding brain function recovery is more difficult in cardiac than in central nervous system disease, hence extra care should be exercised.

The authors consider that the neurologic findings necessary for the determination of cerebral death may be divided into absolute and incidental. A diagnosis of cerebral death may be based on absolute criteria only, in case all of them are proved. The incidental criteria must be supplemented by other findings.

*Absolute criteria* (1) Loss of consciousness, profound coma. (2) Bilateral dilated and fixed pupils. No oculo-spinal reflex. (3) Loss of muscle tone of the extremities and trunk. No postural activity. (4) No change in heart rate by test of carotid sinus reflex and oculo-cardiac reflex. (5) Non filling of cerebral arteries in bilateral carotid and vertebral angiography. (6) No swallowing, yawning, or speech.

*Incidental criteria* (1) Absence of body temperature regulation may constitute important evidence of cerebral death. (2) A marked decrease in the difference in the oxygen saturation of the carotid artery and jugular vein. (3) The presence of the reflex of pupal automatism does not exclude cerebral death. Reaction to pain stimuli to the upper and lower extremities, especially the latter, should not be considered as part of the reflex. The presence or absence of reaction to pain stimuli should be determined from the supraorbital nerve, a branch of the trigeminal nerve, at the supraorbital foramen. (4) Cerebral death cannot be ruled out even if one or more superficial or deep reflexes remain, for instance the persistence of the tendon reflexes (especially those of the upper extremities), cremasteric reflex and bulbocavernosus reflex cannot exclude cerebral death. (5) Cerebral death may have occurred even if anal sphincter tone persist.

The EEG is important in the final assessment of cerebral death. A careful and somewhat elaborate technique is essential. (1) Each EEG recording is made continuously for 20 minutes until an isoelectric EEG (Vh) appears. The EEG is mixed into the isoelectric EEG. (2) The EEG recording is repeated twice or three times and the isoelectric EEG observed at 6 hours after its initial appearance. (3) The EEG recording is made at full gain or at three fold sensitivity, an isoelectric EEG will however appear. (4) Light stimuli, pain stimuli, electro-stimulation of the peripheral nerves and the intracarotid injection of activating agents produce no changes in the isoelectric EEG. (5) Calibration is

frequently repeated with an interelectrode resistance under  $50\,000\ \Omega$ . Recording is made both unipolarly and bipolarly with a paper running speed of under  $3\text{ cm/s}$ . When a EMG becomes mixed in an FEG flaxedil is administered and the recording repeated. Interference always arises from outside or from bodily movements. Two monitoring non cephalic electrodes are placed on the back of the right hand. The ECG and respiratory curves are recorded simultaneously.

The changes in the brain stem that produce cerebral death may be divided into those that affect respiration and those that influence blood pressure.

*Respiration* (1) Ordinary breathing ceases and artificial respiration is necessary (over 24 hours continuously). (2) With artificial respiration stopped for 3 to 5 minutes ordinary breathing does not reoccur. (3) Artificial respiration performed for 10 minutes with room air produced no recurrence of ordinary breathing. Further frequent blood  $pO_2$   $pCO_2$  measurements are desirable.

*Blood pressure* (1) With no change in the ECG or no cause for any change in the circulatory blood volume such as loss of blood present an abrupt fall in blood pressure is important in determining the point of loss of function of the brain stem. As far as the recognition of the drop is concerned since it is clinically difficult to let nature take its course and to observe the depression down to minimal values vasopressor agents are given when the fall reaches  $70\text{ mm}$ . As the precise value may not be recorded the abruptness should be considered as important. (2) The raising and maintenance of blood pressure is possible by the administration of vasopressor agents to produce peripheral contraction.

Death in human subjects should be assessed and certified by qualified observers. It must be emphasized that the determination of cerebral death is quite different from that indicated by cardiac arrest. It thus becomes desirable to call in the assistance of neurosurgeons, neurologists or those specializing in cerebral death in addition to those who treated the patient to assess the case and issue a report.

## SUMMARY

Cardiac transplantation has focussed attention upon the necessity of the accurate determination of the point of death. The investigation proved the significance of loss of brain stem function in the assessment of cerebral death which should now be accepted as the only true indication of death of the body. The criteria of cerebral death especially those concerned with a fall in blood pressure and arrest of ordinary breathing are discussed in detail.

## ZUSAMMENFASSUNG

Die Herztransplantation hat die Aufmerksamkeit auf die Notwendigkeit den Todeszeitpunkt genau zu bestimmen gerichtet. Die Untersuchung zeigt die Bedeutung des Verlustes

der Hirnstammfunktion als Kriterium des cerebralen Todes die jetzt als das einzig richtige Zeichen des körperlichen Todes akzeptiert werden sollte. Die Kriterien des cerebralen Todes besonders im Hinblick auf den Blutdruck fall und den Stillstand der normalen Atmung werden im einzelnen besprochen.

## RÉSUMÉ

La transplantation cardiaque attire l'attention sur la nécessité de déterminer de façon précise le moment de la mort. Ce travail a montré l'intérêt de la disparition des fonctions du tronc cérébral pour déterminer la mort cérébrale qui devrait maintenant être considérée comme le seul signe véritable de mort du corps. Les auteurs examinent en détail les critères de mort cérébrale spécialement ceux qui concernent la chute de la pression sanguine et l'arrêt de la respiration spontanée.

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AUS DER NEUROCHIRURGISCHEN UNIVERSITÄTSKLINIK (DIREKTOR PROF. H. KRAUS),  
DER INTENSIVBEHANDLUNGSTATION (DIREKTOR PROF. I. STEINBEREITHNER),  
DER CHIRURGISCHEN UNIVERSITÄTSKLINIK (DIREKTOR PROF. P. FUCHSIG) UND  
DEM INSTITUT FÜR ANÄSTHESIOLOGIE (DIREKTOR PROF. O. MAYRHOFER), WIEN,  
ÖSTERREICH

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## CEREBRALER ZIRKULATIONSTILLSTAND IN KORRELATION MIT EEG UND pO<sub>2</sub>-AVD- UNTERSUCHUNGEN

von

G. Pendl, J. A. Ganglbauer, I. Steinbereithner und Ch. Tschakaloff

Der Wunsch verlässliche Kriterien zur Feststellung einer irreversiblen Hirnschädigung bei Schädel-Hirntraumen bzw. intrakraniellen Massenblutungen und akuten raumfordernden Prozessen anderer Genese möglichst frühzeitig herauszuarbeiten ist durch die jüngste Entwicklung auf dem Gebiete der Organtransplantation geradezu eine Notwendigkeit geworden (PENN & KAUFER). Auf Grund der weitgespannten Problematik und des Urteilscharakters der Diagnose Hirntod wurden auf internationaler Ebene die erforderlichen Voraussetzungen festgelegt, über die wir schon an anderer Stelle kurz berichtet haben und auf die daher nicht näher eingegangen werden soll. Nun aber lassen sich in einzelnen Fällen Abweichungen im Verhalten von einem oder mehreren der als typisch anzusehenden Parameter feststellen, womit die Diagnose Hirntod scheinbar in Frage gestellt wird. Aus einer Serie von 62 nach allen Parametern synchron durchuntersuchten Fällen stellen wir 5 Fälle vor, die anfänglich durch die oben erwähnte mangelnde Korrelation der Befunde auffielen. Ist dieser Pro-

zentsatz gegenüber Fällen von nicht bestimmbarem Hirntod auch gering oder erscheinen und diese Ergebnisse doch von Bedeutung, da man erwartungsgemäss immer wieder auf solche Fälle stossen wird.

### Kasuistik

*Fall 1* Kompressionsfraktur links frontal, Schädeldarmfraktur 20 Uhr 20. Klinik Spontanatmung bei Mittellirneinklemmung, Angiographie, Verlangsamte Zirkulation ohne Seitenverschiebung EEG Dysrhythmisch AVID Art 305 B jug links 19, 9 Uhr 10 — Klinik Atemstillstand und Zeichen des Hirntodes, Angiographie, Zirkulationsstopp bds EEG Abgeflachte Theta Delta (ab 15 Uhr isoelektrisch) AVID Art 62 B jug links 28 B jug rechts 38 0 Uhr 30. Klinik Atemstillstand und Zeichen des Hirntodes, Exitus 10 min später EEG Isoelektrisch AVID Art 75 B jug links 77 B jug rechts 69.

*Fall 2* Schweres Schädelhirntrauma 13 Uhr 30. Klinik Keine Spontanatmung, bei Zeichen des Hirntodes, Angiographie, Zirkulationsstopp bds EEG Flache Theta Delta mit gelegentlich eingestreuten Beta AVID Art 31 B jug links 22 B jug rechts 29 0 Uhr 30. Klinik Keine Spontanatmung, bei Zeichen des Hirntodes, Angiographie, Zirkulationsstopp bds EEG Flache Wellen AVID Art 27 B jug links 96 B jug rechts 19 1 Uhr 30. Klinik Keine Spontanatmung, bei Zeichen des Hirntodes EEG Isoelektrisch.

Es handelt sich um zwei Fälle mit vollständigem Zirkulationsstopp bei beidseitiger Carotisingiographie. Gleichzeitig wurden EEG Untersuchungen und eine Bestimmung der  $pO_2$  AVID durchgeführt. Dabei stellte sich heraus, daß der Zirkulationsstillstand im supratentoriellen Bereich zwar prognostische Aspekte aufdeckt, die Diagnose Hirntod allein auf Grund des vorhandenen Hirnkreislaufstillstandes noch nicht gestellt werden kann, da EEG und  $pO_2$  AVID in diesen beiden Fällen noch auf eine Cerebralfunktion hinweisen.

*Fall 3* Schädelhirntrauma mit hypoxischem Hirntod 9 Uhr 40. Klinik Spontanatmung bei weiten, lichtstarren Pupillen EEG Nicht registriert AVID Art 380 B jug links 44 11 Uhr. Klinik Atemstillstand und Zeichen des Hirntodes, Angiographie, Verlängerte Zirkulationszeit, keine Seitenverschiebung EEG Isoelektrisch AVID Art 105 B jug links 100 17 Uhr — Klinik Atemstillstand und Zeichen des Hirntodes EEG Isoelektrisch.

Das Auffallende bei diesem Falle ist, daß bei isoelektrischem EEG und fehlen der  $pO_2$  AVID bei Angiographie eine Zirkulation festzustellen ist, d. h. es kommt trotz vorhandener Durchblutung zu keiner Utilisation des Sauerstoffs. Das Sinken der corticalen Funktion ist durch ein isoelektrisches EEG untermauert.

*Fall 4* Apoplektischer Hirnstromtumor mit Mittellirneinklemmung 13 Uhr — Klinik Atemstillstand und Zeichen des Hirntodes, Angiographie, Bds normale Füllung bei Zeichen des Hydrocephalus EEG Isoelektrisch mit gelegentlich generalisierten synchronen Burst steiler Wellen 15 Uhr 15. Klinik Atemstillstand und Zeichen des Hirntodes EEG Isoelektrisch AVID Art 72 B jug links 29 III Uhr 20. Klinik Atemstillstand und Zeichen des Hirntodes EEG Isoelektrisch AVID Art 75 B jug links 41 20 Uhr 15 — Klinik Herzstillstand EEG Isoelektrisch AVID Art 77 B jug links 38.



Angiographischer Nachweis einer Externa Drainage über den Sinus transversus in den Bulbus jugularis bei Kompressionsstillstand der A. carotis interna

Hier handelt es sich um eine isolierte Schädigung des Mittelhirn Hirnstammbezuges, die durch einen apoplektischen Tumor hervorgerufen wurde.

Die Entwicklungszeit des klinischen Hirntodes beträgt zwei Stunden nach Eintritt der Apoplexie.

Da das Gebiet der A. carotis interna außerhalb der Schädigung lag, ergab sich abgesehen von einer hydrocephal ausgespannten A. pericallosa eine völlig normale Füllung. Aus demselben Grund ist der Sauerstoffverbrauch und somit die AVD des Grosshirns im Rahmen der Norm. Es zeigt sich in diesem Falle, daß trotz vorhandener Großhirndurchblutung und normalem Sauerstoffverbrauch ein funktionell totaler Zusammenbruch der Gesamthirnfunktion eingetreten ist, was aus der klinisch neurologischen Untersuchung und dem über sechs Stunden stummen EFG ersichtlich ist.

Fall 5: Enzephalitis (autopsisch verifiziert) 11 Uhr — Klinik: Atemstillstand bei Mittelhirneinklemmung. EEG: Nicht registriert. AVD: Keine. 14 Uhr — Klinik: Atemstillstand bei Mittelhirneinklemmung (Probetrepantation bds., massiver Hirndruck). Angiographie bds.: Zirkulationsstopp mit Externadrainage über den Sinus transversus. AVD: Art. 330 B. jug. links 335 B. jug. rechts 125. 16 Uhr — Klinik: Zeichen des Hirntodes. EEG: Isoelektrisch. 19 Uhr — Klinik: Herzstillstand. EEG: Isoelektrisch.

Bei diesem akuten Ereignis nach generalisiertem Anfall mit nachfolgendem Atemstillstand bildete sich trotz klinischer, angiographischer und EEG-mässiger Zeichen des Hirntodes noch eine Sauerstoff-*AVD* heraus. Die anatomische Realität einer nachgewiesenen Drainage des Externakreislaufes über den Sinus transversus in den Bulbus jugularis gibt eine ausreichende Erklärung für den Widerspruch der Befunde (siehe Abbildung).

### Diskussion

Die Beurteilung der Hirnfunktion des Schwerverletzten respektive die Beurteilung der Diagnose Hirntod, dient in erster Linie der rechtzeitigen Bestimmung des Zeitpunktes einer Organentnahme aber auch für die Entscheidung hinsichtlich Beendigung von Reanimationsmaßnahmen. Nach den internationalen Richtlinien wird das synchrone Bestehen aller für die Feststellung der irreversiblen Hirnschädigung alle als wesentlich angesehenen Parameter gefordert. Die mitgeteilten Befunde lassen aber erkennen, daß in manchen Fällen zwar einzelne Parameter die völlig infauste Prognose eindeutig beweisen (SPANN et coll.), andere Kriterien jedoch abweichende, scheinbar widersprüchliche Befunde ergeben, die für das Bestehen einer gewissen Hirnfunktionen zu sprechen scheinen.

Wenn wir im eigenem Arbeitsbereich bisher stets zugewartet haben, bis alle geforderten Kriterien erfüllt waren, so scheinen unsere Beobachtungen doch dafür zu sprechen, daß die klinische Beurteilung im Verein mit anderen für die infauste Prognose sprechenden Parametern im Interesse des Organempfängers (STEINBEREITNER) eine Vorverlegung des Beginns der sogenannten Schwebephase rechtfertigen könnte.

### ZUSAMMENFASSUNG

Es wird über einige Fälle berichtet, an denen zum Teil bei vollständigen Zirkulationsstop bei beidseitiger Carotisangiographie gleichzeitig EEG Untersuchungen sowie Bestimmungen der  $pO_2$  AVD durchgeführt wurden. Bei einem Zirkulationsstillstand im supratentoriellen Bereich können zwar prognostische Aspekte aufgedeckt werden, die Diagnose Hirntod jedoch nicht mit Sicherheit gestellt werden. In einem Fall bestand trotz angiographisch vorhandener Zirkulation des Gehirns ein iso-elektrisches EEG sowie Fehlen der  $pO_2$  AVD.

### SUMMARY

Electroencephalography and estimations of the  $pO_2$  AVD were undertaken in several cases of arrested circulation during bilateral cerebral angiography. With such arrest in the supratentorial region these methods permitted limited predictions but did not necessarily signify that cerebral death had occurred. Angiographic circulation was demonstrable in one case with an iso-electric EEG and no  $pO_2$  AVD factor could be recorded.

### RÉSUMÉ

Les auteurs présentent quelques cas dans lesquels l'angiographie carotidienne bilatérale avait montré un arrêt circulatoire complet et au cours desquels on a pratiqué simultanément des examens électro-encéphalographiques ainsi que des mesures de la différence artérioveineuse de la  $pO_2$ . Au cours de l'arrêt circulatoire dans le territoire sustentorial on peut découvrir des éléments de pronostic; mais on ne peut cependant pas poser avec certitude le diagnostic de mort cérébrale. Dans un cas il y avait malgré une persistance angiographique de la circulation cérébrale un EEG iso-électrique ainsi qu'une absence de différence artérioveineuse de  $pO_2$ .

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## ARRET CIRCULATOIRE INTRACRANIEU DANS LA MORT DU CERVEAU

Angiographie avec injection sous pression

par

B VLAHOVITCH P FREREBEAU A KUIENER B STOPAK B ALLAIS et C GROS

De nombreux travaux (13, 15, 20, 24, 26, 28, 29, 34 37 38 39) parus pour certains depuis plus de 10 ans montrent bien l'interet accorde a cet etat plus proche de la mort que de la vie constitue par le coma depasse. Des variantes cliniques ayant la meme signification generale sont venues completer la definition initiale etablie en 1959 par MOLLARET & GOULOU (6 11 17 22, 25 26, 28 33, 41). C'est ainsi que la perte fonctionnelle du systeme nerveux intracranien ou « mort du cerveau » est acceptee par la grande majorite des auteurs comme synonyme de mort de l'individu. Cet etat de mort du cerveau laisse subsister souvent une activite fonctionnelle de la moelle epiniere. La persistance des reflexes medullaires dorso-lombaires est la regle en particulier la flexion lente du gros orteil a la stimulation plantaire du pied. Les reflexes medullaires hauts tres elabores sont plus rares et fugaces ils surviennent dans 1/10 des cas (8 17, 18 26 33).

Aux criteres cliniques et electro-encephalographiques deja bien etablis, l'angiographie est venue apporter a retardement avec la notion d'arret circulatoire, un argument de grande valeur pour le diagnostic precis et rapide de mort du cerveau (8 12 17 19 26 33 39). La frequence de mort du cerveau dans notre service de neuro-chirurgie est d'environ 50 cas par an.

Le but de notre travail est de montrer que l'aspect negatif connu jusqu'a present sous le terme de « non visualisation des arteres intra-craniennes » (a l'angiographie de routine ou a la gamma angiographie) pouvait etre transforme en aspect positif de « visualisation circulatoire intra crânienne » grace a l'angiographie faite sous pression avec injecteur

Cette methode permet de concretiser l'arret circulatoire de deceler la permeabilite des arteres leur position et egalement de constater les fuites du produit de contraste par voie retrograde, anastomotique (ophthalmique et carotide externe) et au quelquefois par les veines et les sinus

### La non visualisation des arteres cerebrales a l'angiographie

En 1953 RUSHEDE & ETHELBERG ont les premiers attire l'attention sur les comas graves avec non visualisation des arteres intra craniennes a l'angiographie carotidienne

En 1959 par une etude clinique et experimentale chez le chat nous avons demontre que l'arret circulatoire etait la consequence du conflit entre l'hypertension intra cranienne et la tension arterielle systemique (15)

Cet arret interesse aussi bien le territoire des arteres carotides que des arteres vertebrales (14 15) Depuis cette etude a ete poursuivie aussi bien sur le plan clinique qu'experimental avec l'appoint des mesures isotopiques du debit cerebral (10 12 13)

Des 1960 on a rapporte dans les cas de coma depasse que les rares angiographies carotidiennes pratiquées, revelaient toutes un arret circulatoire cerebral (39)

Les criteres angiographiques ont pris progressivement une grande valeur diagnostique par l'accumulation des examens pratiques dans les cas de mort du cerveau

L'arret circulatoire cerebral doit etre global, carotides et vertebrales. En effet d'authentiques arrêts carotidiens peuvent coexister avec une circulation vertebrale conservee (12 14 15) et inversement la circulation vertebrale peut etre arretee sans arret carotidien

*Les niveaux d'arrêts angiographiques* Ils sont assez variables pour la carotide 3 niveaux peuvent exister arret extra-crânien arret au viphon le plus frequentement au dela du polygone de Willis avec injection de la carotide opposee et exceptionnellement au dela du polygone jusqu'aux premiers centimetres de l'artere sylvienne ou de l'artere cerebrale anterieure Pour l'artere vertebrale, il peut exister egalement 2 niveaux d'arret l'un extra-crânien et l'autre a l'origine du tronc basilaire avec impregnation de l'artere vertebrale opposee



Fig 1 Arrêt circulatoire au niveau du siphon carotidien par injection manuelle

Dans chaque cas la circulation dans le territoire de la carotide externe bien que ralentie s'effectue normalement et progresse jusqu'aux veines épi-crâniennes qui deviennent visibles. Les collatérales meningeées sont exceptionnellement vues.

*Images observées dans la sertiographie* Chaque fois dans le déroulement angiographique le produit de contraste persiste jusqu'à plusieurs minutes dans la carotide avec dans les temps tardifs les images « significatives » décrites sous le nom de « Waterfall » dans le siphon carotidien (29) (Fig 1). On peut également mettre en évidence pour l'artère vertébrale la même image de « Waterfall » grâce à l'angiographie carotidienne sous pression (Fig 2).

Dans aucun cas il n'y a d'impregnation veineuse intra crânienne.

*Les preuves de l'arrêt circulatoire* Le déroulement angiographique dans les cas de mort du cerveau démontre avec conviction l'arrêt circulatoire presque total. Le contrôle par méthodes isotopiques de l'arrêt circulatoire a été effectué par différentes techniques. La première consiste à réaliser une gamma angiographie par injection de  $^{133}\text{Xe}$  dans la carotide interne. Le débit observé (Fig 3) est effondré à 5 ml/100 g/min et exceptionnellement à 10 ml/100 g/min (10, 13, 16, 17). Quelquefois on observe un pic initial de passage intra vasculaire qui est bref et fugace du probablement à une fuite anastomotique artérielle à la base du crâne.

D'autres auteurs utilisant la clearance isotopique ont pratiqué des techniques un peu différentes : injection carotidienne avec blocage rétrograde du vaisseau pour favoriser le passage du produit dans le crâne avec le même résultat (5). Également on a voulu apporter l'isotope localement dans le cerveau par trepano ponction (15) avec les mêmes résultats.

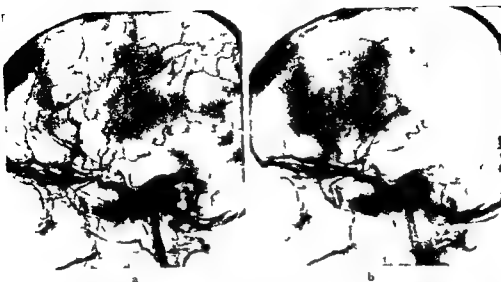


Fig 2 Angiographie carotidienne par injection sous pression a) Temps precoce b) 20 secondes apres Remplissage arteriel cerebral Visualisation du reseau ophtalmique et disparition de la portion intermediaire en (b) Arret vertebreal

L'emploi de la gamma angiographie avec camera a scintillation demontre clairement l'arret circulatoire encephalique par la non visualisation des ombres vasculaires arterielles et veineuses intra-craniennes (11)

### La visualisation des arteres cerebrales a l'angiographie

Dans ce travail ont ete analyse les resultats obtenus au cours de 51 angiographies par injection sous pression pratiquees dans un lot de 25 cas de mort du cerveau

Chaque cas presentait les criteres cliniques et electro-encephalographiques de mort cerebrale De plus chaque fois une angiographie prealable par injection manuelle demontrait la non visualisation des arteres cerebrales.

*Angiographie par injection sous pression* Dans un premier temps l'angiographie sous pression a ete pratiquee chez le chien normal en utilisant un catheter en teflon intra carotidien l'artere etant libre des 2 cotes Une injection de 10 ml de produit de contraste triode (chien de 20 kg) sous 3 1/2 kg de pression revel un excellent remplissage intra et extra-cranien avec une duree totale du temps arteriel de 5 secondes L'injection chez le meme chien peut etre pratiquee jusque 4 fois (total de 2 ml/kg) L'animal reste normal au reveil

Chez l'homme en coma depasse on catheterise l'artere carotide interne avec un trocart mousse en teflon de 1 mm de diametre Une seringue de 30 ml et

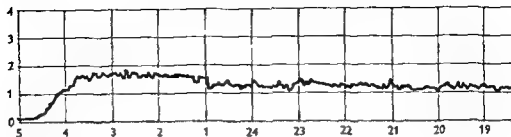


Fig 3 Courbe de débit cérébral carotidien par clearance isotopique dans un coma dépassé  
Débit moyen gris 1 31 blanc 3 82 et total 5 13 ml/100 g/min

contenant 20 à 30 ml de contraste triode d'utilisation courante est adaptée à un injecteur. La pression utile pour le remplissage des artères intra-crâniennes varie entre 2 1/2 et 3 1/2 kg. À noter que l'injection à la main donne une pression de 1 1/2 à 2 kg à la seringue.

AMUNDSEN a employé récemment dans un cas une injection sous 8 kg de pression.

Lorsqu'on veut injecter l'artère vertébrale (rarement nécessaire car dans 40 pour-cent des cas les artères vertébrales peuvent se remplir par voie carotidienne grâce à la fuite anastomotique du produit) il faut utiliser le cathétérisme par sonde de Seldinger et injection de produit de contraste directement dans l'artère vertébrale d'abord à la main puis à l'injecteur.

Les injections sous pression par voie humérale rétrograde sont rarement efficaces même sous très forte pression (4 et 5 kg) pour donner un remplissage vertébral ou carotidien intra-crânien.

La prise des clichés se fait avec les incidences de face et de profil. La durée de la sériographie dans nos 25 cas a varié entre 20 et 60 secondes, 17 cas entre 2 et 31 minutes. Il cas.

**Le remplissage artériel.** Dans tous les cas on obtient dès la fin de l'injection la visualisation des branches de l'artère carotide interne homo-latérale jusqu'aux artéioles sub-millimétriques. Mais bien souvent les 2 systèmes carotidiens s'anastomosent. Dans 40 pour-cent des cas les 2 artères vertébrales deviennent visibles grâce à la voie anastomotique extra-crânienne ou intra-crânienne (Fig 2).

Enfin dans 72 pour-cent des cas on obtient un remplissage du système ophtalmique avec ses 3 portions: l'artère ophtalmique à l'origine sur 1 1/2 cm la portion intermédiaire anastomotique avec les branches de la carotide externe enfin le réseau choroïdien de la coupole postérieure du globe oculaire. Nous allons voir dans le chapitre suivant que la zone anastomotique avec la circulation carotidienne externe se vide assez rapidement alors qu'aux 2 extrémités du

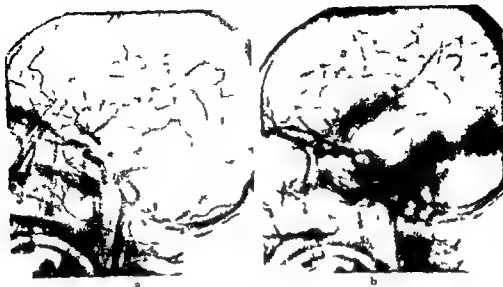


Fig 4 Arrêt circulatoire carotidien par angiographie avec injection sous pression (a) Temps precoce b) 15 minutes après

reseau arteriel ophtalmique il y a stagnation du contraste indiquant un arrêt circulatoire (Fig 2)

*Mobilité et modification du contraste durant la scierographie* En general la visualisation arterielle est maximale des la fin de l'injection Les arterioles intra crâniennes se voient jusqu'aux confins des territoires vasculaires intra crâniens Pendant plusieurs secondes et quelquefois minutes le produit de contraste stagne et l'examen minutieux des extremités des petites branches arterielles montre qu'il n'y a aucune progression identifiable du contraste Cependant, si dans certains cas cet aspect persiste jusqu'à 20 minutes et 30 minutes en general il y a une diminution de l'opacification arterielle avec parfois mobilité du contraste permettant de trouver un sens de progression On a pu ainsi dans ce lot de 25 cas de mort du cerveau trouver 3 variétés principales de remplissage vasculaire intra crânien à l'angiographie sous pression

(1) Les cas d'arrêt total avec stagnation du produit de contraste dans les arteres (Fig 4) Cet arrêt circulatoire total a été observé dans 13 cas (52 pour cent) avec un maximum de durée d'observation de 20 minutes Aucune veine ni sinus ne sont visibles Une fuite du produit de contraste est possible par voie carotidienne retrograde au bout d'un long temps d'observation Dans ces cas d'arrêt total on peut observer un remplissage partiel d'un pedicule intra-cranien qui persiste et change pendant toute la durée de la scierographie

(2) Mouvement du produit de contraste par voie arterielle anastomotique

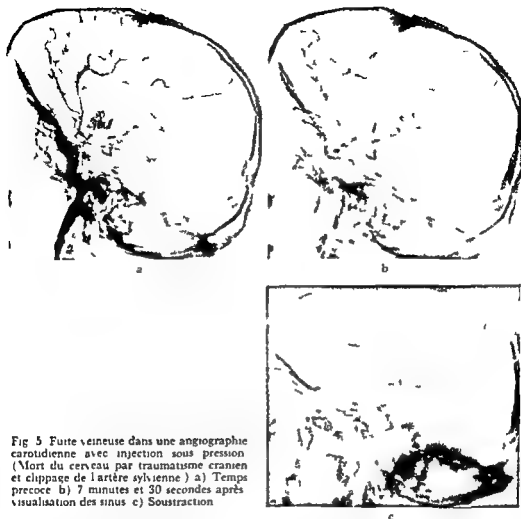


Fig 5 Fuite veineuse dans une angiographie carotidienne avec injection sous pression (Mort du cerveau par traumatisme crânien et clippage de l'artère sylvienne) a) Temps précoce b) 7 minutes et 30 secondes après visualisation des sinus c) Soustraction

Ce mouvement entraîne une diminution nette de la visualisation artérielle intracranienne. Il est possible de déceler d'une façon objective par la sèriographie, les différentes voies de progression ou de fuite du produit de contraste. Celui-ci utilise au minimum 3 voies identifiables avec précision: le polygone de Willis avec passage dans la carotide opposée dans le tronc basilaire et les artères vertébrales; la carotide interne qui se vide par voie rétrograde; ou le système ophthlalmique par sa portion intermédiaire anastomotique avec les branches de la carotide externe qui peut également entraîner le produit.

Ces différentes fuites anastomotiques ont été observées nettement dans 6 cas (24 pour-cent).

(3) Passage artério-veineux et fuite veineuse. Six cas (24 pour cent) entrent dans cette catégorie. Le passage du produit de contraste se identifie dans le sinus longitudinal supérieur ou le sinus latéral. Exceptionnellement il y a visualisation d'une veine corticale. Le sinus latéral s'impregne au bout de 20 à 30 secondes puis il y a une stagnation dans ce sinus pendant plusieurs minutes (plus de 7 minutes dans 1 cas). Cette stagnation coïncide avec l'impregnation des artères (Fig. 5). Parfois comme dans 3 de nos cas (12 pour cent) il y a une impregnation progressive des sinus avec disparition jumelée de la visibilité des artères sur une durée dépassant 60 secondes. Dans tous ces cas les autres critères de coma dépassé (cliniques, EEG et non visualisation à l'angiographie manuelle) étaient présents.

Dans ces derniers cas on peut évoquer non seulement une fuite passive du produit de contraste qui est simplement propulsé dans les veines mais la persistance d'une circulation réduite à un minimum avec passage artério-veineux par vis à tergo.

*Remplissage artériel cérébral et tension artérielle systémique* Il est intéressant de souligner la corrélation qui existe entre le remplissage artériel et la tension artérielle systémique.

Dans les cas de mort du cerveau les sujets sont en général en collapsus vasculaire et catecholamino-dépendants. Depuis longtemps on s'était rendu compte que dans l'arrêt circulatoire par procédé manuel d'angiographie toute tentative d'élever la tension artérielle systémique ne modifiait en rien la situation car cela augmentait parallèlement l'hypertension intra-crânienne par perte de l'autorégulation vasculaire cérébrale et LANGFITT et coll. ont particulièrement insisté sur ce sujet (23).

Dans l'angiographie avec injection sous pression, une hypertension artérielle peut même empêcher le remplissage vasculaire intra-crânien, alors que si la tension artérielle systémique est abaissée, on arrive facilement à visualiser les artères cérébrales.

*Exemple* Mort cérébrale post-traumatique avec persistance d'une réactivité spinale basse. L'électro-encéphalogramme est nul. Une première angiographie avec injection manuelle est faite alors que le patient a 22 de tension maxima. L'injection de la carotide s'arrête à la partie basse du siphon. Une deuxième injection est faite à l'injecteur 30 ml de produit dans une seringue de 50 ml à 3 1/2 kg de pression, alors que la tension artérielle est à 21. L'arrêt se fait au siphon. Une troisième injection est faite dans les mêmes conditions avec une seringue de 30 ml seulement la tension artérielle est toujours à 21. L'arrêt se fait au polygone de Willis (la pression d'injection est plus puissante à cause de la taille réduite de la seringue). Une quatrième injection est faite dans les mêmes conditions avec une tension artérielle à 17. Le polygone de Willis seul est encore visible. Une cinquième injection est faite dans les mêmes conditions mais avec une tension artérielle cette fois-ci entre 6 et 7 de maxima (par réduction de la dose de catecholamine) on obtient un très bon remplissage des artères intra-crâniennes qui sont filiformes.



Cette constatation permet de comprendre aussi que dans certains cas de mort du cerveau avec arrêt circulatoire, l'angiographie manuelle peut arriver à visualiser les artères cérébrales au niveau du polygone et même au delà sur quelques centimètres avec stagnation du produit de contraste. En effet le gradient entre la pression de l'injection (quelle soit manuelle ou mécanique) et la tension artérielle systémique explique la variabilité des niveaux d'arrêt du produit de contraste dans le crâne.

*Remplissage artériel cérébral et pression intra-crânienne* En général dans les arrêts circulatoires cérébraux nous avons toujours trouvé dans les cas explorés une identité entre la tension artérielle systémique et la pression intra-crânienne. Les ventricules et les espaces liquidiens dans un coma dépassé ont habituellement réduits au minimum sauf quand il y a hydrocéphalie ou existence d'un kyste tumoral. Dans ces cas on peut par un drainage ventriculaire ou une ponction du kyste réduire ainsi la pression intra-crânienne.

*Exemple* Dans ce cas de mort cérébrale il existait un hématome de la fosse postérieure situé dans la protubérance. La ponction ventriculaire a été facile à réaliser et la pression trouvée était de 30 cm d'eau alors que la tension artérielle systémique prise dans la carotide était de 2,5 cm de Hg. La pression ventriculaire a été ramenée à 0 par drainage. L'angiographie carotidienne par injection manuelle a pu visualiser le polygone de Willis, la carotide opposée ainsi que l'origine de l'artère cérébrale antérieure sur quelques centimètres avec stagnation du produit de contraste sur toute la scéno-graphie. Une injection sous pression a été ensuite réalisée permettant la visualisation de toute la vascularisation cérébrale sans apparition de veine ni de sinus.

Le remplissage artériel par injection manuelle dans ce cas peut dépendre du gradient de pression entre l'injection et la tension artérielle systémique qui était très basse mais aussi il peut être en rapport avec la diminution de la pression intra-crânienne. En effet dans certains cas publiés (12, 14, 15, 29, 30) de coma dépassé avec tumeur kystique cérébrale et arrêt circulatoire chaque fois la ponction du kyste en diminuant la pression intra-crânienne a permis à une nouvelle angiographie par injection manuelle de faire un remplissage artériel plus complet et donner l'illusion d'une recirculation.

*Passage veineux dans la mort du cerveau par les angiographies sous pression* Dans les 25 cas étudiés nous avons obtenu 6 fois la visualisation partielle des sinus ce qui indique le franchissement de l'étape artério-veineuse par le produit de contraste. En séparant ces cas en 2 variétés : simple fuite dans les sinus et présence d'une microcirculation nous avons voulu souligner deux morphotypes angiographiques.

Il n'est pas exclu que dans les autres cas il n'y ait pas un résidu de circulation dont la démonstration n'est pas faite faute de preuves valables. Même les

mesures isotopiques du débit résiduel ne sont pas convaincantes à cause des difficultés techniques d'introduction du traceur et aussi de la sensibilité de la méthode pour des passages aussi minimes. Dans un cas de mort cérébrale avec arrêt circulatoire angiographique une étude per opératoire de la circulation en employant la technique des cathétérismes d'artères sub millimétriques (35-36) a été réalisée. Nous avons constaté que si on sectionnait une petite veine corticale il apparaît dans le bout proximal de cette veine un très discret filet de sang indiquant clairement qu'il y a un résidu de circulation avec passage artério-veineux. De même en injectant du serum physiologique dans la direction antérograde d'une artère sub millimétrique dans ce même secteur vasculaire cérébral le mince filet de sang sortant par la veine est transformé en écoulement. Ces constatations expliquent que l'arrêt circulatoire cérébral dans ce cas de mort du cerveau, n'était pas un arrêt complet du passage artério-veineux. Il faut remarquer que le large volet de trépanation pratiqué dans ce cas a permis d'éliminer la composante d'hypertension intra crânienne.

D'ailleurs dans toutes les injections artérielles post mortem on arrive toujours à visualiser les veines avec souvent une effraction du lit vasculaire. Mais toute tentative de réaliser une véritable circulation serait infructueuse comme le prouve l'étude des organes en circulation extra corporelle au stade de dégradation cellulaire (20-32).

L'étude pathologique de la partie terminale de l'arbre vasculaire cérébral a montré à certains (2-7) qu'il y avait obstruction mécanique d'un grand pourcentage de petits vaisseaux mais pas de tous ce qui peut expliquer la possibilité d'un passage artério-veineux par l'angiographie sous pression.

### Discussion

Pour établir un diagnostic de mort du cerveau, il est indispensable de s'entourer du maximum de garanties formelles. En effet cet état laisse subsister les autres organes vivants et quelquefois une activité réflexe spinale qui est riche et spectaculaire surtout dans sa forme haute. Les critères cliniques électro-encéphalographiques et angiographiques dans la mort du cerveau pris isolément, présentent tous des insuffisances plus ou moins grandes qui méritent d'être connues. L'ensemble de ces critères dans un même cas donne cependant la plus grande sécurité pour un diagnostic précis et rapide.

*Les critères cliniques.* L'activité totale du système nerveux n'a pas en elle-même une valeur absolue surtout dans certaines étologies toxiques de coma ou en hypothermie (12-26-27).

Il existe quelquefois des situations encore plus trompeuses ou une activité encéphalique totale avec apnée et libération des réflexes médullaires (présence d'un

reflexe cutané plantaire en flexion lente) peut se transformer momentanément en coma avec respiration conservée

*Exemple* Coma métabolique grave avec progressivement apnée. En quelques heures apparition de signes de libération médullaire basse avec réflexe cutané plantaire en flexion lente et réflexe cremastérien. La tension artérielle est basse 7 cm de Hg.

Alors qu'on se préparait à faire une angiographie carotidienne par injection manuelle et à augmenter la tension artérielle par des catecholamines le malade lutte brusquement contre le respirateur et reprend un automatisme respiratoire. L'angiographie n'est pas faite le malade reste dans cet état pendant une heure et demi et redevient comateux aréactif avec apnée mais cette fois-ci tous les autres critères électro-encéphalographiques et angiographiques démontrent la mort du cerveau.

Cet exemple montre avec d'autres déjà publiés (12, 13) que les signes cliniques de la mort du cerveau ne sont pas toujours irréversibles tout au moins au début de leur évolution.

Cela impose dans ces cas d'adopter la plus grande prudence d'interprétation.

*Les critères électro-encéphalographiques* De nombreux travaux montrent la grande importance et les limites de l'électro-encéphalogramme dans le diagnostic de la mort cérébrale. Notamment pour la durée nécessaire d'observation d'un électro-encéphalogramme nul qui même dépassant plusieurs jours peut ne pas être suffisant dans certaines conditions étiologiques comme les intoxications (1, 3, 4, 5, 9, 21, 26, 27, 31, 40).

Les enregistrements profonds donnent les mêmes résultats (12, 38). Il faut cependant savoir que les nerfs crâniens eux-mêmes sont capables de donner une réponse évidente à la stimulation dans les cas authentiques de mort du cerveau.

*Les critères angiographiques* L'arrêt circulatoire encéphalique est un signe éminemment convalidant de mort cérébrale à condition d'en connaître quelques aspects particuliers.

(1) Le problème de la réversibilité de l'arrêt circulatoire. Certains auteurs (14, 29, 30) ont écrit que l'arrêt circulatoire pouvait être quelquefois réversible. Mais tous les exemples cités restent discutables soit qu'il s'agit de simple remplissage artériel après une ponction de kyste tumoral (14, 29, 30) soit que l'arrêt carotidien (une seule artère explorée) ait été observé chez des malades avec respiration conservée (29, 30) donc non en coma dépassé. Dans ces cas une reprise de la circulation carotidienne peut se produire.

(2) Les arrêts circulatoires partiels. Ils peuvent être carotidiens ou vertébraux. On a déjà écrit des cas de comas graves avec mydriase bilatérale et aréflexie cornéenne mais sans apnée qui présentaient un arrêt circulatoire carotidien bilatéral mais avec circulation vertébrale conservée (12, 18, 34). Ces cas ont tous en commun à l'examen clinique des mouvements respiratoires une arcac

tivité totale mais au si quelquefois des signes de rigidité de decerebration sans jamais presenter une liberation des reflexes medullaires

Inversement il peut exister des cas et nous montrerons un exemple ou le tableau clinique est exactement celui d'une mort cerebrale mais ou la circulation carotidienne est presente mais ralentie alors que la circulation vertebrale est arretee

*Exemple* Traumatisme cranien avec impact temporo occipital droit et intervalle libre de 12 heures Ensuite s'installe un coma areactif avec apnee et presence d'un automatisme medullaire bas En effet on decelait un reflexe de flexion lente du gros orteil et des reflexes rotuliens et achilleens avec triple retrait

L'electroencephalogramme revelait un trace plat avec artefacts d'activite cardiaque mais quelques rares bouffees courtes d'ondes lentes et une activite musculaire temporale ne permettaient pas de conclure a un trace nul

L'angiographie carotidienne manuelle a montre une circulation conservee avec temps arteriel durant 6 à 7 secondes et temps veineux au dela de la 10ieme seconde Il n'y avait aucun engagement

Une tentative d'injection vertebrale par catheterisme avec sonde de Seldinger du cote gauche echoue de visualiser le tronc de la vertebrale Une angiographie humerale retrograde droite visualise la carotide intra craniennne mais montre un arret extra-cranien de l'artere vertebrale On pouvait donc evoker dans ce cas qu'il y avait un arret de la circulation vertebrale

Une nouvelle angiographie faite 2 heures plus tard a revele un arret de la circulation carotidienne et vertebrale

L'autopsie a permis chez ce malade de trouver un hematome extra dural de la fosse posterieure avec ramollissement complet du cervelet du bulbe et de la protuberance

Dans cet exemple on remarque que le tableau clinique peut etre celui d'une mort cerebrale avec arret circulatoire partiel dans la fosse posterieure sans arret carotidien L'electroencephalogramme dans cet arret circulatoire partiel n'était pas absolument nul L'evolution du malade s'est faite vers un arret circulatoire complet

Il s'agit exactement de la contre partie des formes avec arret carotidien et circulation vertebrale conservee

Ces quelques aspects de l'angiographie dans les comas depasses prouvent que l'arret circulatoire n'est pas uniquement la consequence d'un conflit hypertension intra crâniennne et tension arterielle de perfusion En effet il est possible d'avoir une hypertension intra craniennne legerement differente suivant les compartiments intra craniens mais cela n'est pas suffisant pour arreter la circulation mecaniquement dans un territoire et pas dans l'autre La degradation cellulaire locale avec la perte de l'autoregulation vasculaire cerebrale permet de mieux expliquer l'arret circulatoire partiel Les travaux experimentaux demontrent que l'arret circulatoire encephalique devient definitif irreversible au dela de 15 minutes (22)

Dans ces conditions, le critère d'arrêt angiographique est primordial pour le diagnostic de mort du cerveau.

Dans la mort du cerveau il ne peut y avoir persistance d'une circulation conservée comme le prouvent tous les documents observés et en particulier l'évolution des cas d'arrêt circulatoire partiel supra ou infratentorial qui aboutissent finalement à un arrêt circulatoire complet. D'ailleurs l'étude des conditions de survie des organes perfusés *in vitro* montre que la mort cellulaire entraîne toujours une augmentation de la résistance vasculaire et un arrêt circulatoire.

### Conclusion

Parmi les différents critères cliniques, électro-encéphalographiques et angiographiques de la mort du cerveau l'arrêt circulatoire est primordial. L'angiographie avec injection sous pression dans les cas de mort du cerveau s'est révélée utile pour de multiples raisons.

Elle permet de visualiser les artères intra-crâniennes et de constater la stagnation du produit de contraste pendant de nombreuses minutes (jusqu'à 20 minutes) ce qui équivaut à un arrêt circulatoire définitif. Cette manière de pratiquer l'angiographie même par l'artère carotide seule peut visualiser souvent les 4 pédicules cérébraux montrant que l'arrêt circulatoire est total. Ce fait est très important car les arrêts circulatoires partiels carotidiens ou vertébraux peuvent être des sources d'erreurs d'interprétation graves.

D'un point de vue physiopathologique vasculaire l'angiographie sous pression montre également d'une façon convaincante que le produit de contraste ne stagne pas indéfiniment dans le crâne mais qu'il y a de nombreuses fuites. Ces fuites se font par voie artérielle rétrograde et anastomotique mais aussi (au minimum dans 24 pour-cent des cas) par passage dans les veines et les sinus. Ce dernier point oblige à accepter que l'arrêt circulatoire n'est pas toujours absolu. La circulation résiduelle si minime soit-elle implique un passage artério-veineux difficile à mettre clairement en évidence par d'autres méthodes (Lötters).

La cause de ce ralentissement extrême de la circulation dans la mort du cerveau est en partie mécanique (conflit entre hypertension intra-crânienne et pression de perfusion) mais aussi elle est en rapport avec la perte de l'intégrité fonctionnelle (autoregulation) de l'arbre vasculaire cérébral et quelquefois de son intégrité anatomique (possibilité d'obstruction vraie du réseau capillaire terminal).

Il ne peut y avoir mort du cerveau ni d'un autre organe avec une circulation conservée.

Si le terme arrêt circulatoire n'est pas littéralement exact, sa signification garde toute sa valeur étant donné que le ralentissement prolonge du temps artériel enlève toute efficacité à la fonction circulatoire.

## RÉSUMÉ

Dans les cas de mort du cerveau avec arrêt circulatoire angiographique l'angiographie carotidienne sous pression (2 1/2 à 3 1/2 kg) permet de visualiser toutes les artères cérébrales (carotide et vertébrale). Le produit de contraste stagne dans les artères de 4 à 20 minutes mais des fuites de ce produit se font vers l'artère ophtalmique et la carotide externe et aussi dans 25 pour cent des cas dans les sinus. Il ne peut y avoir mort du cerveau ni d'un autre organe sans arrêt circulatoire local.

## SUMMARY

Carotid angiography at a pressure of 2.5 to 3.5 kg will fill all the carotid and vertebral cerebral arteries in cases of cerebral death in which the ordinary angiographic circulation has ceased. The contrast medium stagnates in the arteries for 4 to 20 minutes but also migrates in the direction of the ophthalmic and external carotid arteries and in 25 per cent of the cases towards the venous sinuses as well. Cerebral death like that of any other organ, means the complete arrest of the local circulation.

## ZUSAMMENFASSUNG

In Fällen von Gehirntod mit angiographischem Zirkulationsstillstand zeigt sich bei der Karotisangiographie unter Druck (2,5 bis 3,5 kg) dass alle Gehirnarterien (Aa. carotis und vertebralis) gefüllt werden. Das Kontrastmittel stagniert in den Arterien während 4 bis 20 Minuten aber es wandert auch in Richtung der Aa. ophthalmica und carotis externa und in 25 Prozent der Fälle in die venösen Sinusse. Weder der Gehirntod noch der Tod eines anderen Organes kann erfolgen ohne Stillstand der lokalen Zirkulation.

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## PNEUMOGRAPHY



## ETUDE TOMOENCEPHALOGRAPHIQUE DES ATROPHIES CEREBELLEUSES

par

J BORIS II FREDI et J ROSIER

C'est sans aucun doute dans l'étude de la fosse postérieure que la tomographie a le plus apporté à l'encephalographie gazeuse et cette technique permet aujourd'hui un diagnostic extrêmement précis des atrophies cerebelleuses. THIEBAUT et coll ont été parmi les premiers à préconiser la tomographie dans l'exploration du cervelet et ces auteurs ont présenté avec SUBIRANA au 7ème symposium neuroradiologicum un travail sur la stratigraphie sagittale médiane dans les atrophies du vermis cerebelleux. A ce même symposium LEFMAN & ABRAHONOWICZ ont montré la grande importance dans le diagnostic des atrophies cerebelleuses de l'image d'élargissement des sillons sur la tomographie sagittale médiane.

Mais, ces différents auteurs n'ont utilisé que la tomographie de profil. Ils paraissent ne s'être intéressés qu'aux atrophies vermineuses. De plus ils ne se sont pas occupés de l'état du tronc cérébral dont l'atrophie accompagne pourtant si souvent celle du cervelet.

Les progrès récents de la tomoencephalographie permettent aujourd'hui une étude extrêmement précise des différents étages du tronc cérébral de face comme de profil et nous disposons maintenant à la Salpêtrière d'un nombre assez im-

portant d'atrophies cérébelleuses explorées de façon systématique à l'aide de tomo-graphies de face et de profil. Il nous a paru intéressant de reprendre le problème du diagnostic radiologique des atrophies cérébelleuses à partir de ce matériel en essayant de confronter nos résultats encéphalotomographiques aux travaux anatomiques de ces quinze ou vingt dernières années.

Ce travail ne s'est pas révélé extrêmement nouveau. THOMAS écrivait en 1932 à propos des atrophies cérébelleuses que c'était là un des chapitres les plus embrouillés de la pathologie cérébelleuse. Il ne fait aucun doute que la première impression qui se dégage de l'étude de la littérature est que cette remarque de THOMAS est toujours valable et que l'on pourrait même dire qu'il s'agit d'un des chapitres les plus embrouillés de la neurologie. Des travaux importants ont été faits depuis 1932, en particulier par l'École Française de Neurologie et DR RICHONDO (1964) ESCOFFIER & MASSON (1967) ont proposé une classification anatomoclinique qui nous a été des plus utiles au cours de ce travail.

**Technique.** Nous ne décrivons pas ici la technique tomoencéphalographique que nous utilisons à la Salpêtrière et qui a fait l'objet d'un autre travail. Rappelons seulement que l'exploration d'une atrophie cérébelleuse doit à notre avis comprendre obligatoirement (1) une zonographie de profil passant par le plan sagittal médian, (2) des coupes tomographiques de face faites tous les centimètres depuis un plan passant 3 cm en avant du conduit auditif externe jusqu'à un plan passant 1 cm en arrière de ce conduit et réalisées la ligne orbitoméridale de Reid inclinée de 30 à 35° sur l'horizontale; ces coupes permettent une excellente étude du tronc cérébral. (3) Nous y ajoutons parfois 2 coupes de face faites ligne de Reid inclinée de 50 à 55° sur l'horizontale et passant 0,5 à 1 cm en avant du conduit auditif externe; ces coupes donnent en effet une excellente image de face du cervelet et en particulier du vermis et de la partie adjacente des lobes.

**Matériel.** Notre matériel comprend 82 atrophies cérébelleuses chez des malades de 2 à 72 ans. Il comprend 55 hommes et 27 femmes; ce qui confirme ce fait bien connu: l'atrophie cérébelleuse est près de deux fois plus fréquente chez l'homme que chez la femme.

Dans 40 cas (soit près de la moitié de notre matériel), une étiologie a pu être retrouvée — encore qu'il ne soit pas toujours facile d'affirmer la relation de cause à effet entre cette étiologie possible et l'atrophie. Dans 22 cas (soit 27 %), il s'agissait de malades éthyliques notoires; dans 10 cas (12 %), d'une maladie familiale et dans 2 cas de malades porteurs d'un cancer.

Notre matériel comprend, en outre 2 comitialités, 2 occurrences d'une atrophie cérébelleuse à une sclérose en plaques, un diabète et une atrophie post-radiothérapique chez un malade irradié pour glioblastome du tème ventriculaire.



Fig 1 Atrophie vermiennne moderee



Fig 2 Atrophie vermiennne importante Ethyle  
Aspect en fougere Gros 4eme ventricule

Nous decrirons dans un premier temps les principaux signes radiologiques que nous avons pu observer dans notre materiel. Puis dans un deuxieme temps nous verrons comment ces signes etaient groupes et les principaux tableaux radiologiques qui peuvent ainsi etre realises.

## Les principaux signes radiologiques

### La zonographie de profil

*L'atrophie du cer-elet* Elle peut se presenter sous trois aspects differents

1 *L'atrophie vermiennne ou « predominance vermiennne »* En 1929 MATHIEU & BERTRAND ecrivaien-t a propos des atrophies cerebelleuses « Le cer-elet miniature est rare. L'atrophie predomine a la face superieure de l'organe mais frappe es essentiellement les lobes quadrilateres mais elle n'epargne pas le vermis. Nous di-sons meme que l'atrophie vermiennne est plus manifeste que l'atrophie hemispherique ». L'atrophie vermiennne est en effet, la forme de beaucoup la plus frequente d'atrophie cerebelleuse : elle represente 68 cas sur les 82 de notre materiel ou 78 % des cas.

THIEBAUT et coll (1966) distinguent les atrophies vermiennes en trois groupes : (1) Les atrophies vermiennes corticales qu'ils distinguent en atrophies localisees a un lobule (atrophie vermiennne superieure et atrophie vermiennne diffuse) (2) Les atrophies vermiennes cortico-sous-corticales de l'adulte (3) Les atrophies vermiennes cortico-sous-corticales de l'enfant.



Fig 3 Importante atrophie vermiennne avec aspect cliquete du cervelet Ethyle



Fig 4 Enorme dilatation du 4eme ventricule et de la partie adjacente de l'aqueduc Image d'elargissement des sillons au niveau du vermis inferieur Cet aspect est tres rare

Nous ne pensons pas que cette classification puisse être conçue telle que la présentent ces auteurs et nous verrons plus loin qu'il nous paraît préférable de classer les atrophies vermiennes en fonction de l'existence ou de la non existence d'une atrophie associée du tronc cérébral.

Quant à l'atrophie du vermis proprement dit elle peut être plus ou moins importante mais elle se traduit toujours par une accentuation plus ou moins prononcée de l'image des lobules, des lames et des lamelles. Elle prédomine toujours sur la partie antéro-supérieure du cervelet. Lorsqu'elle est peu importante, il y a trop d'air à la partie antéro-supérieure du cervelet, et les lames et lamelles de cette portion du cervelet ont anormalement bien dessinées (Fig 1). Lorsqu'elle est très importante elle prédomine toujours à la partie antéro-supérieure du cervelet de sorte que celui-ci n'est pas atrophié de façon égale suivant tous les diamètres et prend un aspect disharmonique. Les sillons entre les lames sont accentués de façon considérable d'où un aspect cliquete en feuille de fougère de la partie supérieure du cervelet (Fig 2, 3, 14a). Quant à l'atrophie du vermis inférieur il est exceptionnel qu'elle soit directement visible sur la zonographie de profil. Nous ne l'avons observée que dans 3 cas (Fig 4).

2 *L'atrophie à prédominance supérieure sans image des lames et des lamelles*  
Ce deuxième aspect de l'atrophie cérébelleuse est infiniment plus rare que l'atro-



Fig 5



Fig 6

Fig 5 Atrophie à prédominance supérieure sans image des lames et des lamelles (atrophie olivo pontocerebelleuse)

Fig 6 Petit cervelet harmonieux Atrophie type Lejoane et Lhermitte

phie vermineuse puis que nous n'avons observé que dans 7 cas (85 % des cas)

L'atrophie prédomine encore au niveau de la partie antéro-supérieure du cervelet (Fig 5) comme dans le cas de l'atrophie vermineuse mais, il n'y a pas d'accentuation de l'image des lames et des lamelles la face supérieure du cervelet reste lisse

Une explication de cet aspect peut être trouvée dans l'étude anatomoclinique de MATHIEU & BERTRAND. Ces auteurs ont montré en effet, que les lamelles des hémisphères cérébelleux contrairement à celles du vermis avaient un pédicule assez court et qu'elles s'écartaient beaucoup moins l'une de l'autre en cas d'atrophie. Il est ainsi permis de se demander si cette deuxième forme d'atrophie cérébelleuse ne correspond pas à une atrophie à prédominance hémisphérique. Dans les 7 cas où nous l'avons observée, elle faisait partie d'un tableau d'atrophie olivo pontocerebelleuse.

3 *Petit cervelet harmonieux* Le troisième aspect d'atrophie cérébelleuse correspond à la forme qui a été décrite sous le nom de petit cervelet harmonieux ou de cervelet miniature. Cette forme est très rare (7 cas dans notre matériel soit 8,5 % des cas). Son diagnostic est souvent très difficile. Le cervelet est diminué de volume selon tous ses diamètres mais sa forme est respectée et ses pro-





Fig 3 Importante atrophie vermiennne avec aspect decouplé du cervelet Ethyle



Fig 4 Enorme dilatation du 4ème ventricule et de la partie adjacente de l'aqueduc Image d'élargissement des sillons au niveau du vermis inférieur Cet aspect est très rare

Nous ne pensons pas que cette classification puisse être conçue telle que la présentent ces auteurs et nous verrons plus loin qu'il nous paraît préférable de classer les atrophies vermiennes en fonction de l'existence ou de la non existence d'une atrophie associée du tronc cérébral.

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■ *L'atrophie à prédominance supérieure sans image des lames et des lamelles*  
Ce deuxième aspect de l'atrophie cérébelleuse est infiniment plus rare que l'atro-



Fig 8

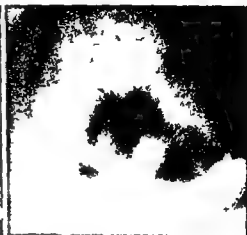


Fig 9

Fig 8 Image d'atrophie vermiennne de face Atrophie corticale tardive de Marie

Fig 9 Importante atrophie du tronc cerebral avec niveaux liquides dans les cisternes de l'angle Atrophie olivo-pontocerebelleuse (forme familiale de Menzel)

*Autres signes mis en evidence par la tomographie de profil* La dilatation du 4eme ventricule est un signe extremement frequent (30 cas dans notre materiel) Il traduit une atrophie de la substance blanche Cette dilatation peut etre extremement importante (Fig 7a) Les parois du ventricule prennent alors un aspect convexe et il s'y associe une dilatation de la partie adjacente de l'aqueduc

La dilatation de la grande citerne est un signe parfois difficile a apprecier du fait de l'aspect tres variable de cette citerne a l'etat normal bien etudie par LILJEQUIST on sait, en particulier qu'elle peut monter a l'etat normal jusqu'a la tente du cervelet

Dans les 15 cas de notre materiel ou cette citerne avait les dimensions les plus importantes on comptait 11 atrophies vermiennes et sur ces 11 cas, le 4eme ventricule etait normal 4 fois ce qui permet de penser que l'atrophie etait surtout corticale On peut donc dire que la dilatation de la grande citerne n'est pas un signe d'atrophie sous-corticale comme le pensent certains auteurs mais beaucoup plus probablement un signe d'atrophie du vermis inferieur

L'elargissement de la citerne pontique et de la citerne interpedonculaire est un signe important d'atrophie du tronc cerebral (Fig 7b)

L'atrophie des tubercules quadrijumeaux est mise en evidence par un elargissement de la citerne de Galien montrant un aspect aplati rabote des tubercules quadrijumeaux Cet elargissement est peu frequente (ou difficile a apprecier) Nous n'en avons observee que 2 cas

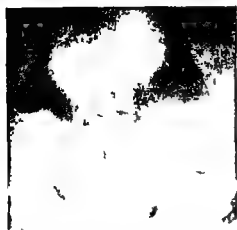


Fig 10 Atrophie du pedoncule cérébral supérieur et du pedoncule cérébral moyen gauche



Fig 11 Atrophie du bulbe Atrophie olivo-pontique bilatérale (forme familiale de Menzel)

### Tomographies de face

Les tomographies de face peuvent mettre en évidence l'atrophie du cercelet. On a fait des coupes ligne orbitomittale de Reid inclinée de 50 à 55° sur l'horizontale. Ces coupes mettent bien en évidence en effet l'atrophie vermineuse et celle de la partie adjacente des hémisphères cérébelleux (Fig 8).

Les coupes de face ont surtout importance pour étudier le tronc cérébral. L'atrophie du tronc cérébral est en effet extrêmement fréquente (38 cas sur les 82 de notre matériel) soit 46 % des cas. Elle se traduit sur les coupes de face (Fig 9, 14 b) par le volume anormalement petit du mésencéphale ou de la protubérance. L'élargissement parfois considérable des cisternes circumponculaires ou tectopontiques. L'élargissement des cisternes de l'angle pontocérébelleux parfois (normale avec souvent alors existence d'un niveau liquide de chaque côté) (Fig 9).

Les pedoncules cérébelleux supérieurs ont touchés dans un nombre important de cas (20 sur 82 soit 20 % des cas de notre matériel) (Fig 10). Leur atrophie se traduit par un aspect anormalement grêle du pedoncule atrophié, avec élargissement de la partie adjacente de la cistère ambiante. Cette atrophie est souvent unilatérale ou à prédominance unilatérale (12 cas sur 20).

Les pedoncules cérébelleux moyens peuvent également être atrophiés le plus souvent de façon unilatérale ce qui leur donne alors un aspect asymétrique sur la tomographie (Fig 10). Cette atrophie paraît peu fréquente, puisque nous ne l'avons observée que dans 5 cas.

Au niveau du bulbe la citerne laterobulbaire peut être très élargie. L'aspect concave en dehors du contour antéro-externe du bulbe est tout à fait anormal, et traduit, selon toute vraisemblance, une atrophie de l'olive bulbaire (Fig. 11).

L'association d'une atrophie cérébrale à une atrophie cérébelleuse est extrêmement fréquente. 36 des 82 cas de notre matériel. Dans 5 cas, une myélographie gazeuse montre l'existence d'une atrophie médullaire associée.

### Les grands tableaux radiologiques

Le groupement des signes radiologiques permet de distinguer deux grands types d'atrophies cérébelleuses selon que l'atrophie du cervelet est isolée ou associée à une atrophie du tronc cérébral. Mais l'aspect différent du cervelet nous amène à distinguer ses atrophies isolées en deux formes de fréquence extrêmement inégale. Enfin, nous étudierons à part le problème des atrophies des peduncules cérébelleux supérieurs. L'étude de notre matériel nous amène ainsi à distinguer quatre grands tableaux radiologiques.

*Premier tableau — L'atrophie vermienne isolée.* C'est la forme de beaucoup la plus fréquente. Elle représente en effet 40 des 82 cas de notre matériel, soit près de 50 % des cas (Fig. 2, 3, 8).

Dans certains cas, une étiologie peut lui être reconnue. C'est le tableau en particulier qui est réalisé par l'atrophie éthylique qui représente 22 des 40 cas d'atrophie vermienne isolée de notre matériel, soit un peu plus de 50 % des cas. Dans deux cas, il s'agissait d'une comitatalité, dont un cas où la comitatalité était secondaire à une anoxie néonatale chez un sujet jeune. Dans deux cas, enfin, l'atrophie vermienne a été reconnue chez des sujets atteints d'une sclérose en plaque évérée.

Dans certains cas rares, il s'agit d'une maladie familiale. C'est alors évidemment l'atrophie corticale familiale de Gordon Holmes. Notre matériel n'en comprend qu'un cas.

Dans un nombre important de cas, enfin, aucune étiologie n'est reconnue, et l'on peut penser alors que le tableau radiologique correspond à l'atrophie tardive à évolution lente de Marie Foix et Alajouanine.

L'aspect radiologique (comme d'ailleurs l'aspect anatomique) est absolument le même dans tous les cas. La seule distinction possible peut être assez artificielle, repose sur l'existence ou la non existence d'une étiologie.

*Deuxième tableau — le petit cervelet miniature isolé.* Il s'agit encore d'une atrophie isolée du cervelet. Mais elle mérite qu'on en fasse un tableau à cause de son aspect particulier et de son extrême rareté. Notre matériel en comprend 3 cas vérifiés.



Fig. 1. Dégenérescence juno-cerebelleux type Marie. Petit cervelet harmonieux et atrophie médullaire. a) Le petit cervelet harmonieux. b) L'atrophie médullaire.

Le premier, chez un enfant de 2 ans et 4 mois, était un cas de l'exceptionnelle atrophie de la couche des grans décrite par NORMAN en 1940 et qui est une maladie familiale primitive. Notre petite malade avait une sœur de 5 ans, que nous n'avons pas radiographiée atteinte de la même maladie.

Dans les deux autres cas, il s'agissait de la non moins exceptionnelle atrophie paraincospasique dont le premier cas a été rapporté par BROUWER en 1919.

Dans l'un de ces deux cas, il s'agissait d'une femme dont le syndrome cérébelleux s'était installé progressivement après une intervention pour cancer de l'ovaire.

Dans l'autre cas, il s'agissait d'un homme dont le syndrome cérébelleux était associé à des brûlures œsophagiennes. L'examen radiologique de l'œsophage n'avait pas mis en évidence de cancer mais à la vérification on trouva un épithélioma œsophagien histologique.

En définitive l'aspect du petit cervelet harmonieux, si on le paraît correspondre au type anatomique décrit sous le nom d'atrophie corticale diffuse.



Fig 13 Atrophie olivo-pontocérébelleuse. Forme sporadique de Dejerine et Thomas. Enorme dilatation du 4ème ventricule et atrophie cérébelleuse extrêmement importante.



Fig 14 Atrophie olivo-pontocérébelleuse. Forme familiale de Menzel. Importante atrophie vermineuse. Gros 4ème ventricule. Importante atrophie du tronc avec dilatation des citernes latéropontiques et des citernes de l'angle pontocérébelleux. Ni eaux liquides dans ces dernières.

*Troisième tableau — L'atrophie du cervelet associée à une atrophie du tronc cérébral.* Elle est extrêmement fréquente puisqu'elle représente 38 des 82 cas de notre matériel, soit presque autant que l'atrophie vermineuse seule.

Elle s'accompagne presque toujours d'une dilatation importante du 4ème ventricule. 5 cas sur 38 seulement avaient un 4ème ventricule normal.

Il n'est pas toujours facile de mettre une étiquette précise sur ces formes. L'aspect du cervelet, l'existence ou la non-existence d'une atrophie associée de la moelle, l'importance de l'atrophie du tronc cérébral permettent cependant, dans un nombre non négligeable de cas, de reconnaître le type anatomoclinique.

Dans 4 cas de notre matériel l'atrophie cérébelleuse était du type petit cervelet harmonieux. Dans un cas associée avec atrophie importante du tronc cérébral, il s'agissait de cette forme exceptionnelle décrite en 1909 par LEJONNE et LHERMITTE sous le nom d'atrophie olivo-rubro-cérébelleuse qui correspond à un ramollissement de la commissure de Werneckh (Fig 6). Dans les trois autres cas le petit cervelet harmonieux était associé non seulement à une atrophie du tronc cérébral (sans dilatation du 4ème ventricule) mais également à une atrophie médullaire. Il s'agissait cliniquement dans les deux cas d'une dégénérescence spinocérébelleuse type Pierre Marie (Fig 12).

Dans tous les autres cas il s'agissait d'une atrophie cérébelleuse à prédominance supérieure. Plusieurs éventualités ont alors à considérer.

Dans certains cas cette atrophie cérébelleuse s'accompagne d'une atrophie importante du tronc cérébral sans atrophie de la moelle. Il s'agit alors presque à coup sûr d'une atrophie olivo-pontocérébelleuse dans sa forme sporadique, type Dejerne Thomas (Fig 7). C'est là certainement l'une des formes les plus importantes des atrophies cérébelleuses (25 cas de notre matériel). L'atrophie cérébelleuse peut être considérable (Fig 13). C'est à ce groupe qu'appartiennent les 7 atrophies cérébelleuses à prédominance supérieure mais sans image de sillon dont nous avons parlé plus haut.

Dans d'autres cas il y a une atrophie médullaire associée. Le problème est alors plus difficile. Il peut s'agir en effet d'une maladie de Friedreich qui s'accompagne d'une atteinte des cellules de Purkinje à prédominance vermienne. Mais il peut s'agir également de la forme familiale décrite par Menzel de l'atrophie olivo-pontocérébelleuse qui s'accompagne de lésions médullaires de même type que celles de la maladie de Friedreich.

La distinction radiologique entre les deux formes est extrêmement difficile.

Il semble cependant que dans l'atrophie olivo-pontocérébelleuse type Menzel l'atrophie du tronc cérébral et du cervelet soient très importantes et de même type que dans la forme sporadique de Dejerne et Thomas avec toujours une dilatation importante du 4ème ventricule (Fig 9-11-13).

Dans la maladie de Friedreich au contraire il semble que l'atrophie du tronc cérébral soit moins importante et le 4ème ventricule normal (Fig 15-16).

*Quatrième tableau — L'atrophie du pedoncule cérébelleux supérieur.* Nous avons fait un tableau de l'atrophie du pedoncule cérébelleux supérieur parce que cette atrophie peut être isolée réalisant alors la divergence cérébelleuse myoclonique ou atrophie dentro-rubrique de Ramay Hunt (Fig 17).

Mais en fait cette forme est tout à fait exceptionnelle et nous ne l'avons observée qu'une seule fois dans notre matériel. Dans tous les autres cas l'atrophie du pedoncule cérébelleux supérieur était associée à une des formes que nous avons décrites plus haut. Dans 11 cas, à une atrophie olivo-pontocérébel



Fig 15 Maladie de Friedreich. L'atrophie cerebelleuse et l'atrophie du tronc cerebral sont peu importantes 4ème ventricule normal Atrophie medullaire



Fig 16 Maladie de Friedreich. Autre cas. Atrophie cerebelleuse modérée. L'atrophie du tronc n'est pas visible 4ème ventricule normal

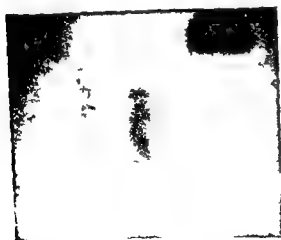


Fig 17 Maladie de Ramsay Hunt. Atrophie isolée du pedoncule cerebelleux supérieur droit

leuse dans 3 cas a une atrophie vermienne type Marie Foix et Alajouanine, dans 4 cas a une atrophie ethylique dans un cas probablement a une lésion cervico spinocerebelleuse type Marie. On sait d'ailleurs que certains auteurs comme Christophe et Gruner pensent que « le syndrome de Ramsay Hunt mérite d'être individualisé » cause des secousses myocloniques et du type de l'atteinte



cerebelleux qui le caractérisent (noyau dentelé et pedoncule cerebelleux superieur) ce syndrome doit être en fait, integre dans le vaste ensemble des maladies degeneratives

### Conclusion

La tomoencephalographie permet une etude extremement precise des atrophies cerebelleuses et permet de diviser celles-ci en quatre grands tableaux radiologiques

(1) *Les atrophies vermiennes isolees* qui realisent une atrophie disharmonieuse a predominance superieure. Tres frequentes elles forment un vaste groupe qui comprend en particulier l'atrophie ethylique l'atrophie familiale de Holmes et l'atrophie tardive de Marie Foix et Alajouanine

(2) Le petit cervelet harmonieux isole, exceptionnel qui traduit une *atrophie corticale diffuse* et peut correspondre soit a l'atrophie de la couche des grains type Norman soit a l'atrophie paraneoplasique

(3) *L'atrophie cerebelleuse associee a une atrophie du tronc cerebral* presque aussi frequente que l'atrophie vermiennne

Si l'atrophie du tronc cerebral est importante avec dilatation du 4eme ventricule et atrophie cerebelleuse a predominance superieure, il s'agit d'une atrophie olivo-pontocerebelleuse soit dans sa forme sporadique de Dejerine et Thomas ou la moelle n'est pas atrophiee soit dans sa forme familiale de Menzel qui s'accompagne d'une atrophie de la moelle. Il existe un cas particulier l'exceptionnelle atrophie olivo-rubrocerebelleuse de Lejonne et Lhermitte ou l'atrophie cerebelleuse parait être du type petit cervelet harmonieux associee a une atrophie du tronc importante

Si l'atrophie du tronc cerebral est peu importante avec 4eme ventricule normal et atrophie medullaire associee c'est une degenerescence spino-cerebelleuse soit maladie de Marie si le cervelet est du type petit cervelet harmonieux, soit maladie de Friedreich si l'atrophie est a predominance vermiennne

(4) *L'atrophie des pedoncules cerebelleux superieurs* enfin est rarement isolee. Il s'agit alors de l'exceptionnelle atrophie dento-rubrique de Ramsay Hunt.

### RÉSUMÉ

La tomoencephalographie permet un diagnostic extremement precis des atrophies cerebelleuses et permet de diviser celles-ci en quatre grands tableaux radiologiques. Il est possible non seulement de reconnaitre l'atrophie du cervelet mais de reconnaitre le type anatomopathologique de cette atrophie

# SUMMARY

Tomoencephalography produces extremely accurate diagnosis of cerebellar atrophy and enables the condition to be divided into four main roentgenologic types. The atrophy may thus be recognized and its pathologic nature and anatomic site determined.

# ZUSAMMENFASUNG

Die Tomoencephalographie gibt eine ausserordentlich genaue Diagnose der Atrophie des Kleinhirns und ermöglicht eine Aufteilung dieses Zustands in vier wesentliche roentgenologische Gruppen. Die Atrophie kann dadurch erkannt werden und dessen pathologische Natur und anatomischer Umfang bestimmt werden.

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## FOURTH VENTRICLE

### III Intra and extra axial tumours

by

MARIO CORRALES

Following the pioneering work of LASHOLM (1939-1946), LINDGREN (1950), and BULL (1950) the problems in the diagnosis of posterior fossa expanding lesions have been those of distinguishing between intra- and extra-axial tumours and fixing their limits (CASTELLANO & RUGGIERO 1953, FALK 1953, LILIEQUIST 1959). The differential diagnosis may usually be reached from the cisternal changes and the size of the lesion is best determined from the displacement of the fourth ventricle (LILIEQUIST 1959). Nevertheless the exact location, the origin and growth of the tumour often still remain to be assessed. This investigation was therefore carried out to see whether a detailed analysis of the changes in the fourth ventricle and its recesses would be helpful. The anatomy and encephalographic appearances of the normal fourth ventricle as well as changes in the latter in tumours of the cerebellum have been described in earlier publications (CORRALES & GREITZ 1972 a, b).

*Definitions* The directions and locations in the posterior fossa were the same as in the previous paper (CORRALES & GREITZ 1972 b). All locations and displacements were consequently oriented in the following directions: anteriorly and

Table 1

*Displacements and deformities produced in the balloon experiments in fig. 1. The classification of tumour sites according to CORRALES & GREITZ (1972). Symbols 1 = present 0 = absent or absence of changes + = increased - = decreased Parenthesized symbols indicate subtle changes*

Balloon tumour number	1	2	3
Site	B4	B4 ex	Clivus
<b>Fourth ventricle</b>			
General displacement	(ant)		
	lat	post	
	(sup)	lat	post
Height	0	0	-
Floor width	-	0	+
Floor tilt	>	>	0
<b>Velum</b>			
Displacement	lat	lat	post
Tilt	>	>	0
Width	0	0	+
Curve frontal	+*	0	-
Curve lateral	+	0	-
<b>Fastigium</b>			
Displacement	sup	post	post
	lat	lat	opened
Compression	(<)	0	0
<b>Nodulus</b>			
Displacement	lat	lat	post
Compression	(<)	0	(<)
<b>Sup post recess</b>			
<b>Sup post recess</b>			
<b>Ipsilateral</b>			
Displacement	post	post	post
	med	med	lat
Compression	>	>	0
<b>Contralateral</b>			
Displacement	0	0	post
			lat
Compression	(<)	0	0
<b>Lateral recess</b>			
<b>Ipsilateral</b>			
Displacement	med	med	lat
	ant	post	
Compression	>	>	>
<b>Contralateral</b>			
Displacement	lat	lat	lat
Compression	0	0	>

with large balloon

Table 2

*Site and pathologic diagnosis of tumours with displacements and deformities. Classification, abbreviations and symbols as in table 1*

Tumour number	1	2	3	4	5	6
Main site	B†	B†	B†	B†	B†	B†
	extra	extra	extra	extra	extra	extra
	axial	axial	axial	axial	axial	axial
Additional component	Pontine	Cl	Pontine	Pontine	Cerebellum	Pontine
Type of tumour	Meningioma	Meningioma	Neurinoma	Neurinoma	Neurinoma	Neurinoma
<b>Fourth ventricle</b>						
General displacement	post lat	post lat	post lat	post lat	post lat	(post) lat
Height	0	0	0	0	0	0
Floor width	+	+	—	(—)	—	+
Floor tilt						<
Velum						
Displacement	lat	lat	lat	(lat)	(lat)	lat
Tilt			>			
Width	0	—	(—)	(—)	(—)	(—)
Curve frontal	0	(+)	—	(+)	(+)	(+)
Curve lateral	0	0	0	0	0	(+)
Fastigium						
Displacement	lat	lat	lat	(lat)	(lat)	(lat)
Compression	0	(>)	0	0	0	0
Nodulus						
Displacement	lat	(lat)	lat	lat	(lat)	(lat)
Compression	0	(>)	0	(>)	( )	(>)
Sup post recess						
Ipsilateral						
Displacement	post	post	post	post	post	post
Compression	0	0	0	>		0
Contralateral						
Displacement	0	0	0	0	(lat)	(lat)
Compression	0	0	0	0	0	0
Lateral recess						
Ipsilateral						
Displacement	post med	post med	post med	post med	post med	post (lat)
Compression	>	>	>	>	>	>
Contralateral						
Displacement	0	lat	0	lat	lat	lat
Compression	0	0	0	>	0	>

in its upper part • less than fastigium

Table 2 (cont.)

7	8	9	10	11	12
Pons	Pons	Medulla oblongata	Quadrig plate	Pituitary region	Mid brain small
0	0	Hydrocephalus	3rd vent splenium	3rd vent mid brain	0
Glioma	Glioma	Not specified	Glioma	Glioma	Glioma
post	post	post	inf	inf	0
—	—	sup	post*	post**	0
+	+	+	0	0	0
0	0	0	0	0	0
post	post	post	inf	inf	0
0	0	0	0	0	0
+	+	+	+	0	0
—	—	—	—	0	0
—	—	—	+	+	0
post	post	post	inf	inf	0
opened	opened	opened	×	×	0
post	post	post	inf	inf	0
inf	inf	inf	×	{ }	0
×			fr above	fr above	
post	post	post	(inf)**	(inf)**	0
0	0	0	0	0	0
post	post	post	(inf)**	(inf)*	0
0	0	0	0	0	0
lat	lat	lat	0	0	0
×	×	×	0	0	0
fr above	fr above	fr med and below			
lat	lat	lat	0	0	0
×	×	×	0	0	0
fr above	fr above	fr med and below			



posteriorly both perpendicular to the clivus and at right angles to these, i.e. superiorly and inferiorly. The cerebellum was divided into five sagittal sections: one medial which included the vermis called section A, and four lateral sections: one medial and one lateral on each side which included the hemispheres, called B and C. Section B was divided into four quadrants: one of which was anterior and inferior and close to the pontine angle; this section was called B4. Brain stem tumours are located inferiorly in the medulla or superiorly in the anterior (pons) or posterior (quadrigeminal plate) part of the brain stem. Extra axial tumours may be located in the midline superiorly or inferiorly, more laterally in the pontine angle although sometimes more inferiorly close to the jugular foramen. Extra axial tumours of the cerebellopontine angle may grow into the anterior inferior part of the cerebellar hemisphere that is into the B4 location.

### Material and Methods

*Autopsy investigation of experimentally produced balloon tumours* The technique of investigating displacements and deformities of the fourth ventricle with a balloon catheter technique and central ventriculography with a water soluble contrast medium was described in a previous paper (CORRALES & GREITZ 1972 b). This technique was employed to demonstrate the difference between intra- and extra axial pontine angle tumours with regard to deformity of the fourth ventricle. In addition the changes produced by an experimental clivus tumour were determined (Table 1).

*Anatomic investigations of tumour material* Twelve specimens of the cerebellum and the brain stem from the museum were examined. This material included 2 pontine tumours, 3 neoplasms of the quadrigeminal plate, 6 extra axial tumours of the cerebellopontine angle and 1 tumour of the medulla oblongata (Table 2). The findings in tumours of the cerebellar pontine angle were compared with those in intracerebellar growths in the B4 position. These specimens were carefully dissected, sectioned and photographed.

*Radiologic investigation of clinical material* Observations of minor anatomic details of the fourth ventricle such as the superior posterior recess and especially the lateral recess were of the utmost importance in the determination of the size of a tumour in the anterior compartment of the posterior fossa. Tomography is often necessary to demonstrate these structures. Only the material examined at Karolinska Sjukhuset was therefore used and only those cases with satisfactory filling of the fourth ventricle were chosen as well as those in which a good idea of the location and size of the tumour could be obtained from the operation report. The material so selected included 51 tumours: 11 intrapontine tumours

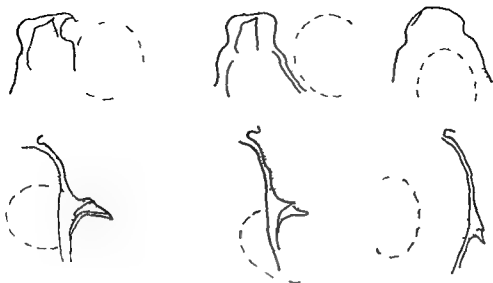


Fig. 1. Displacements produced in experimental intra- and extra-axial balloon tumours. Site B4 ex (left), B4 ic (middle) and pons (right).

3 tumours of the quadrigeminal plate, 8 medullary tumours and 32 tumours of the pontine angle. Among the latter tumours were 22 verified and 4 non-verified neurinomas, 2 meningiomas, 1 chordoma, 2 cholesteatomas and 1 glomus jugulare tumour, all examined by tomography with a linear tomograph Mimer II (FREDZELL et coll. 1968). Three acoustic neuromas were bilateral. All 4 non-verified neurinomas were associated with dilatation of the internal meatus.

### Results

*Ventriculographic and autopsy investigation of experimentally provoked tumours.* Particular attention was paid to the deformity of the fourth ventricle produced by a tumour in the pontine angle, with particular emphasis on the differentiation between intra- and extracerebellar neoplasms. Experimental tumours were therefore produced in these two positions. The deformities caused by an extra-axial balloon tumour on the clivus were also investigated (Table 1). This latter tumour produced approximately the same deformity as occurred with intra-pontine growths in the pathologic and clinical material. Tumours in the angle caused different deformities of the fourth ventricle depending upon whether their location was intra- or extracerebellar. When the balloon tumour



Fig 3 Autopsy material a) Intracerebellar medulloblastoma in the B4 position with lateral compression of the body of the fourth ventricle and narrowing of its untiled floor ( $\rightarrow$ ) The velum ( $\rightarrow$ ) is tilted posteriorly on the tumour side b) Extra axial meningioma in pontine angle The fourth ventricle is compressed from the anterior aspect its floor ( $\rightarrow$ ) is widened on the affected side and tilted posteriorly as is the velum ( $\rightarrow$ ) c) Intracerebellar medulloblastoma in the B4 position with posterior displacement of the posterior superior recess ( $\rightarrow$ ) which is also displaced posteriorly in (d) by the deformity caused by an extra axial neurinoma The compression of the brain stem ( $\rightarrow$ ) is most marked posteriorly in (c) and anteriorly in (d) (e) Intracerebellar haemorrhage

was intracerebellar the fourth ventricle was compressed from the lateral aspect the velum but not the floor tilted posteriorly and the superior posterior recess pushed posteriorly and compressed from the lateral aspect The lateral recess was displaced medially in its posterior part and anteriorly (Fig 1) The extra cerebellar balloon tilted the roof including the velum, the fastigium and the posterior superior recess as well as the floor posteriorly on the side of the tumour The posterior superior recess was displaced posteriorly to a greater extent than the fastigium with both locations but it was not compressed from the lateral aspect by the extra axial tumour The lateral recess was displaced medially in its anterior part and pushed posteriorly

*Anatomic investigation of tumour* Four of the six tumours at the cerebellopontine angle were encroaching mainly upon the pons one being purely intracerebellar The different appearances of the fourth ventricle and its recesses in intra and extracerebellar pontine angle tumours as evident in the balloon experiments were confirmed in the pathologic investigations (Fig 2) In all the extra axial tumours both the floor and velum were tilted posteriorly on the affected side and the width of the floor was increased in those growing into the pons and slightly decreased in the neoplasm growing into the cerebellum (Fig 2 g) The two intracerebellar lesions in the B4 position produced considerable lateral compression of the fourth ventricle with slight tilt of velum and the floor in the opposite directions (Fig 2 a c e) One of these specimens revealed narrowing and an increase in the curve of the velum a change that could be produced experimentally by a large balloon The superior posterior recess in both intra and extra axial growths was displaced posteriorly on the affected side The same differences in the lateral recess were however noted as in the balloon experiments i.e. the lateral recess was pushed posteriorly by the extra axial neoplasms which did not occur with the intracerebellar location The lateral recess could be also pushed medially by an extra axial tumour growing into the cerebellum (Fig 2 g) and occasionally laterally when it extended into the pons (Fig 2 h) The medial displacement of the lateral recess was most marked anteriorly with the extra axial tumours and posteriorly with intracerebellar involvement (Fig 2 c d) No obvious differences were

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displacing the recesses medially the change being more marked in the posterior part of the lateral recess (→) as opposed to the deformity in (f) meningioma where the medial shift of the (posteriorly displaced) lateral recess (→) is more marked anteriorly g) The intracerebellar involvement of this acoustic neuroma caused compression and medial displacement of the lateral recess (→) and brain stem with a concomitant decrease in the width of the fourth ventricle The posterior displacement of the floor (→) distinguishes this lesion from a B4 intracerebellar tumour h) The intra pontine growth of this neurinoma explains the flattening of the brain stem (▷) on the affected side and the lateral and posterior shift of the lateral recess (→) this posterior displacement distinguishes the tumour from non pontine lesions

found as regards the deformities caused by neurinomas and those produced by meningiomas in the pontine angle

Tumours of the pons led to the well known posterior displacement and compression of the fourth ventricle which was always broader than normal. The tonsils were displaced inferiorly but not separated (Figs 3, 4). Due to their inferior displacement the floor of the superior posterior recess was no longer formed by the tonsils, but the white matter of the cerebellum (Fig 4 a). The posterior displacement of the nodulus and that of the velum increased the angle of the fastigium, which was now not pointed, but appeared more rounded (Fig 3). When the tumour was growing in the inferior part of the brain stem, the lateral recesses became displaced laterally and hence separated. They were usually also much compressed and flattened from above so that they appeared broader in the horizontal plane. This broadening might be considerable (Fig 4) the inferior border being formed mainly by the cerebellum and to a lesser degree by the tonsil. The tumour of the medulla oblongata caused a more marked separation of the lateral recesses in relation to the deformity of the fourth ventricle than in the case of pontine tumours (Fig 5). They were compressed from below and broadened in the frontal plane. This broadening, was however less marked than in the pontine neoplasm. As opposed to the findings in pure pontine tumours, the tonsils were separated (Fig 5 b c). Tumours in the upper posterior part of the brain stem, i.e. those originating in the colliculate plate caused quite a different deformity of the fourth ventricle which was displaced inferiorly. Its upper part was also moved posteriorly like in an axial pressure cone (LILLEQUIST 1960). The fastigium was displaced down to the level of the superior posterior recesses much in the same way as with tumours of the upper vermis and appeared more pointed than normal (Fig 6).

*Clinical material* Five of the eight intrapontine gliomas in this material were growing symmetrically in the pons. The cases with the asymmetric growth will be discussed in conjunction with the pontine angle tumours. All eight gliomas caused a broadening of the floor of the fourth ventricle. The height was decreased in five and in three gliomas it was normal due to dilatation of the fourth ventricle. The angle between the velum and the nodulus was more obtuse and the fastigium had a rounded instead of a pointed appearance in all but one of the eight cases (Fig 7). The lateral recesses were displaced laterally and broadened evidently due to compression from the superior aspect (Fig 8). The origin of the lateral recesses was displaced inferiorly in 3 cases. With the exception of the three asymmetric pontine tumours, one of which compressed the posterior part of the fourth ventricle from the lateral aspect the posterior superior recesses were displaced posteriorly to the same extent and hence the fastigium was not tilted, nor was there any tilt of the velum in these 5 cases.



Fig 3 Autopsy material Gliomas of the pons a) The angle of the fastigium is less pointed than normal the curve of the anterior medullary velum everted and the tonsil displaced inferiorly b) Great increase in the angle of the fastigium which is rounded instead of pointed. Marked inferior displacement of the tonsil which takes part in the formation of the floor of the posterior superior recess to a lesser extent than normal. The nodulus is flattened and pushed inferiorly.



Fig 4 Autopsy material Pontine gliomas a) Marked widening of the lateral recess as evident from the increased distance between the restiform body (►) and the indentation at the level of the dentate nucleus (▶). The inferior wall of the lateral recess (↔) and the floor of the posterior superior recess (↗) which is displaced inferiorly but not laterally b) Horizontal cut at a higher level than in (a). The lateral recess (↔) is compressed from the medial and superior aspects and flattened in the sagittal as well as in the horizontal plane.



Fig 5 Autopsy material Medulloblastoma of the medulla oblongata a) Although the tumour protrudes into the inferior part of the fourth ventricle it does not infiltrate the pons which is pushed upwards. The angle of the fastigium is slightly wider than normal ( $\rightarrow$ ) due to the posterior displacement of the nodulus ( $\blacktriangleright$ ) b) Basal view. Separation of the tonsils ( $\triangleright$ ) by the tumour c) A frontal cut demonstrates compression of the lateral recess ( $\leftrightarrow$ ) from the medial and inferior aspects. The tela choroidea ( $\triangleright$ ) forming the floor of the lateral recess is also displaced laterally d) Horizontal cut displaying the lateral recess ( $\leftrightarrow$ ) widened by compression from below and displaced laterally e) The floor of the posterior superior recess is formed mainly by the tumour (T) due to lateral shift of the tonsil and posterior growth of the tumour

Fig 6 Autopsy material. Cloma involving the collicular plate and adjacent structures. The body of the pons is not involved. The fastigium is compressed from above and displaced inferiorly. The curve of the velum is increased. The superior part of the fourth ventricle is raised due to the inferior and posterior displacement of the mid brain.

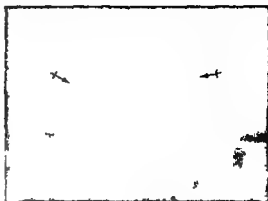


Fig 7 Clinical material. Pontine glioma. a) Fastigium (→) rounded and its angle wider than normal due to the posterior displacement of the velum and nodulus. b) The increased width of the fourth ventricle indicates a greater distance between two posterior superior (→) and lateral recesses (→) and the lateral walls (▴). The tonsils are not separated i.e. the vallecula is not broadened.

Tumours of the medulla displaced the pons superiorly and might therefore protrude into the posterior part of the fourth ventricle without infiltrating the pons (Figs 5-11). The upward displacement of the brain stem produced a kink of the aqueduct (Figs 10-11). There was also separation of the lateral recesses (Figs 9-10-11) which were broadened probably due to compression. The nodulus in these tumours was pushed posteriorly and sometimes superiorly as opposed to the findings in pontine gliomas when it usually was displaced



Fig. 8. Encephalography. Asymmetric pontine glioma with asymmetric compression of the fourth ventricle. The lateral recess ( $\rightarrow$ ) is compressed mainly from the medial aspect on the left side on which the tumour is larger; it is broadened on the right side, probably due to compression from above. The vallicula is displaced to the right, but not widened.



inferiorly. Separation of the tonsils causing a widening of the vallicula was always evident, a change never present in pure pontine tumours. With two exceptions the e.g. growths were more or less asymmetric and the displacement and compression of the posterior superior recesses and of the lateral recesses might be different on the two sides (Figs 10-11), thus asymmetric growth caused a tilt of the velum in two cases.

No midline extra-axial tumours were evident in this material. One clival chordoma was growing laterally in the cerebellopontine angle and will be discussed in this connection.

The three tumours of the quadrigeminal plate produced inferior displacement of the aqueduct, which was kinked at the border between the posterior colliculate body and the fourth ventricle, the anterior part of which was raised due to the posterior displacement of the mid brain (Fig. 12). The inferior displacement of the central lobulus pushed the fastigium inferiorly to the level of superior lateral recesses (Fig. 12). This displacement also caused a broadening of the velum.

Tumours of the cerebellopontine angle may be extra-axial, intra-axial or intracerebellar. Neoplasms of the cerebellum may grow into the pons or vice versa. Extra-axial tumours may extend into the pons or into the cerebellum. It seemed likely that a careful investigation of the fourth ventricle and its recesses would be of importance for the exact localization of the tumour in these cases. An intracerebellar involvement of an extra-axial tumour should cause a deformity similar to that described (CORRALES & GREITZ 1972 b) as being characteristic of an intracerebellar tumour in the B4 position (Fig. 1). An intracerebellar tumour compresses the body of the fourth ventricle from the lateral aspect, the velum is tilted posteriorly on the affected side and the floor — as estimated from the origin of the lateral recesses — is not tilted or is tilted anteriorly on



Fig 9 Clinical material. Astrocytoma of the upper medulla. a) Midline tomographic cut in the sitting position. Enlargement of the medulla, compression of the canal of Magendie, widening of the fast gum (→) and flattening of the nodulus (▶). b) Same case. Supine position with cut more lateral. The origin of the lateral recess (↔) is displaced posteriorly. c) The separation of the lateral recesses (↔) and that of the tonsils (▶) are evident. The posterior continuation of the vallicula embracing the nodulus (▶) is opened up. d) Tomographic cut. Fast gum (↔), posterior superior recesses (→), lateral recesses (↔), vallicula (▶). The widening of the vallicula is a feature not evident in a pure pontine glioma.

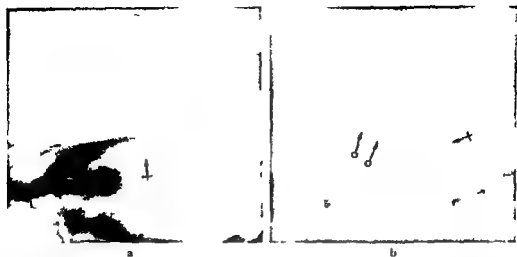


Fig. 10 Asymmetric tumour of the medulla (not verified). The deformity of the inferior part of the floor ( $\rightarrow$ ) on the right side proves the involvement of the medulla. The posterior part of the lateral recess ( $\rightarrow$ ) on the left side is displaced superiorly and compressed from below and therefore broadened in the  $\pi$  projection. Kinking of the aqueduct is caused by upward displacement of the pons.



Fig. 11 Astrocytoma of the medulla verified at operation. a) The large indentation in the inferior part of the floor of the fourth ventricle does not necessarily imply involvement of the pons (see Fig. 3a). The upper brain stem is pushed superiorly producing a kink in the aqueduct. Marked posterior displacement of the tonsil which is delineated posteriorly by the retrotonsular space ( $\rightarrow$ ). b) The asymmetric growth of the tumour produces a tilt of the posterior superior recesses ( $\rightarrow$ ) and of the fastigium ( $\rightarrow$ ).

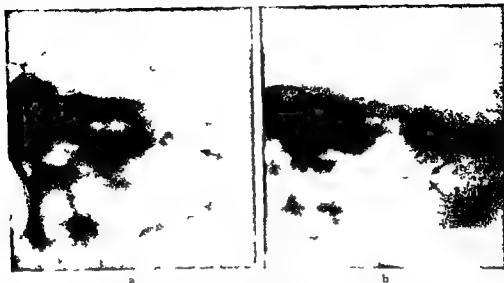


Fig 12 Gliomas of the quadrigeminal plate a) Marked inferior and anterior displacement of the aqueduct produces a kink at the caudal end at the aqueduct ( $\longleftrightarrow$ ). The superior part of the fourth ventricle ( $\rightarrow$ ) is raised due to the inferior and posterior displacement of the mid brain. Fast gum moved inferiorly b) Increased curve of the velum ( $\longleftrightarrow$ ) is apparent together with a decreased angle of the fast gum ( $\rightarrow$ ). The chiasm is displaced down to the level of the posterior superior recesses ( $\longleftrightarrow$ )

the side of the tumour medial and anterior displacement of the lateral recess on the affected side is more marked posteriorly. This latter finding is however opposed to the changes with extracerebellar pontine angle tumours (Figs 13-14) in which like acoustic neuromas the anterior part of the lateral recess was more displaced than the posterior recess (Fig 14). Depending upon the predominance of either the pontine or the intracerebellar component the displacement was sometimes medial, it was sometimes lateral although generally only a posterior shift of the lateral recess was present (Fig 13). This posterior displacement never occurred with an intracerebellar growth. The body including the floor the posterior superior recess and the velum were all displaced posteriorly on the side of the tumour. The posterior superior recess was more compressed in the intracerebellar neoplasm than with an extracerebellar pontine angle tumour. A decreased height of the fourth ventricle was never evident in pontine angle tumours. An increased width was present in all pontine tumours and frequently but not always in pontine angle neoplasms. With intracerebellar invagination an extracerebellar angle tumour produced marked lateral compression of the fourth ventricle (Fig 15).



Fig 10 Asymmetric tumour of the medulla (not verified). The deformity of the inferior part of the floor ( $\rightarrow$ ) on the right side proves the involvement of the medulla. The posterior part of the lateral recess ( $\rightarrow$ ) on the left side is displaced superiorly and compressed from below and therefore broadened in the a.p. projection. Kinking of the aqueduct is caused by upward displacement of the pons



Fig 11 Astrocytoma of the medulla verified at operation. a) The large indentation in the inferior part of the floor of the fourth ventricle does not necessarily imply involvement of the pons (see fig 5a). The upper brain stem is pushed superiorly producing a kink in the aqueduct. Marked posterior displacement of the tonsil which is delineated posteriorly by the retrotonsillar space ( $\rightarrow$ ). b) The asymmetric growth of the tumour produces a tilt of the posterior superior recesses ( $\rightarrow$ ) and of the fastigium ( $\rightarrow$ ).



Fig 15 Left sided acoustic neuroma growing into the cerebellum. Marked compression of the fourth ventricle as distinct from intracerebellar pontine angle tumour. However posterior displacement of the medullary isthmus (▶) and hence of the external opening of the lateral recess is present a feature not dependent on intracerebellar tumours. The posterior superior recess is pushed posteriorly on the side of the growth (→) causing a double contour in the lateral view. The obex (▶) indicates the inferior part of the fourth ventricle.

wurden mit denen die experimentell durch einen Ballonkatheter hervorgerufen waren verglichen. Der Wert der Tomographie bei den encephalographischen Untersuchungen wird betont.

## RÉSUMÉ

L'auteur a étudié les modifications de petits détails anatomiques du quatrième ventricule dans 51 cas de tumeurs intra- et extra-axiales examinées à l'autopsie ou par encéphalographie et ventriculographie. Il a comparé ces modifications à celles qu'il a simulées expérimentalement au moyen de ballonnets. Il souligne l'intérêt de la tomographie dans les examens encephalographiques.

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## NORMAL INTRACRANIAL SUBARACHNOID SPACES IN CHILDREN

An anatomic and radiographic investigation

by

J I EISENMAN L M ROSEN and B J O LOUGHLIN

There is a dearth of information concerning the normal anatomy and radio-graphic appearance of the endocranial subarachnoid spaces (cisterns and sulci) in infants and children. ROBERTSON (1967) indicated that to evaluate encephalograms it is necessary to have a standard of normality. Some generalizations regarding the cisternal structures in children have been made by this author: the chiasmatic and interpeduncular cisterns apparently are deeper in the pediatric patient; the pontine cistern is of much the same depth as in adults; the pontocerebellar cisterns on the other hand are more capacious than in adults. ROBERTSON however did not offer definite criteria of normality.

Recently CRIPSON & LODIN (1969) studied the size of the interpeduncular pontine pontocerebellar and cisterna magna in children. Their work is the first effort to establish normal dimensions and growth patterns of the pediatric cisterns.





Fig 1

Fig 1 Lateral film of cistern injection of neonate body. The barium gelatin mixture in the posterior soft tissues of the neck gives testimony to the difficulty of precise placement and maintenance of the injecting needle in infants and small children.

Fig 2 In this eviscerated body of the neonate the lower lumbar vertebral bodies have been removed. A plastic catheter has been inserted into the subarachnoid space and advanced into the upper cervical region.



Fig 2

The value of encephalography has been well established in the diagnosis of intracranial mass lesions, atrophic lesions, and detection of cerebral herniations (DAVIDOFF & EPSTEIN 1955, AZAMBUJA *et coll* 1956, RUGGIERO 1957, TAVERAS & WOOD 1964). It was not until the last 20 years, with the widespread use of fractional encephalography as advocated by LINDOREN (1957) and ROBERTSON (1967), that the true need for knowledge of the roentgen appearance of the subarachnoid cisterns was realized. By introducing small amounts of gas into the subarachnoid space and not withdrawing any cerebrospinal fluid, the encephalography can be performed despite clinical evidence of increased intracranial pressure. In some instances of intracranial masses, the ventricular system does not fill and subarachnoid cisterns are the only demonstrable air-filled intracranial structures. These so-called failed encephalograms are now considered of definite value because they often provide crucial information as to lateralization of expansive processes, tentorial herniations, and even demarcation of a tumor.

Our knowledge of the roentgen anatomy of the ventricular system of the brain

is more detailed because for many years the introduction of gas into the ventricles was considered a much safer procedure. The subarachnoid cisterns are not demonstrated by this technique.

Reference to encephalography in the pediatric age group is scanty in the world literature. DANDY'S (1918) first publication on encephalography dealt with children of 12 years of age and in a later work 50% of the case material were children. MARTIN & UHLER (1922) reported a series of lumbar encephalographic examinations performed on children and adults with possible tumor. COLLNITZ (1951) described what he called normal child encephalogram. He noted the difficulties associated with encephalography in the lower age groups: restlessness, changing size of the head due to growth, etc. His material was confined to pediatric psychiatric patients; the relationship between encephalography and motor and psychologic development being the primary concern of his investigation. BRENNER (1962) reported the results of investigation of 13 children between 3 months and 3 years of age with apparently normal nervous systems. He found that the smallest infants had a relatively large ventricular system and drew attention to the size of the third ventricle. LILJEQUIST (1959) in his comprehensive work on the roentgen appearance of the normal cisternal structures, did not include pediatric patients. DI CIURO (1966) states that the subarachnoid cisterns in children have a different appearance than in adults and he stresses the necessity of basic anatomic-radiographic work in this field. TAYLOR & WOOD (1964) describe the cisterns in children as being proportionately larger than in adults.

The purpose of this investigation was to evaluate the radiographic limits of position, size and shape of the subarachnoid cisterns in normal newborn infants and children. Pediatric encephalographic examinations, retrospective and prospective, were used as well as a small number of postmortem cases.

### Post mortem investigations

Initial efforts were directed to the cisternal puncture technique for the injection of air and positive contrast material. This technique was used in the 4 to 16 year old age group. Below this age it was too difficult to maintain the needle within the subarachnoid space. Injection into the soft tissues of the neck was not unusual (Fig. 1). Two needles were found to be more efficient for adequate drainage of the cerebrospinal fluid. With the body in the supine position, the needles were walked down from the midline of the inferior occipital bone. The needle point was directed toward the nasion. Needles of 14 to 16 gauge were used. All accessible cerebrospinal fluid was drained. Frontal, cross table lateral and half axial views were exposed. The body was then turned to the prone position.

tion with the head as low as possible and the casting material injected. Care was taken to introduce the material with as little pressure as possible. The barium gelatin, while hot, required the least pressure. The various silicone mixtures required the most pressure.

In the newborn and infant where size did not cause a problem in the handling of the body an injection was made into the spinal subarachnoid space. POTTS et coll (1969) suggested an approach to the subarachnoid space through an incision into the external tissues over the lumbar spine. Our modification of this procedure is to remove the vertebral bodies of L 1, 2 and 3 after examining the vertebra during the postmortem examination. The dura is incised and a polyethylene 205 catheter or a pediatric feeding tube is advanced to the level of the cervical spine. If necessary the catheter is tied at its entrance into the subarachnoid space to seal the space and allow uniform injection (Fig. 2).

### *Casting materials*

**Silicone rubber** Silicone has many of the characteristics of an ideal casting material. COOK & MARGULIS (1962) perfected a technique for its use in casting the lumen of the rectosigmoid colon. Curing (hardening) is not temperature dependent in the range used in cadavers (7° C to 21° C). The rubber is easily cut permitting sectioning of the brain. It is more suitable for casting impressions of surfaces such as cisterns, than for small lumina of blood vessels. The hardened silicone does not shrink even after immersion in formalin. Liquid silicone is used to dilute the mixture and make a less viscous mixture.

The disadvantages are: (1) The viscosity of the silicone is greater than any of the agents used. The required pressure for injection resulted in rupture of the plastic into the brain. (2) The attempts to liquify the silicone result in non uniform hardening.

The most successful formula used is: 50 g Dow Corning Sialastic RTV 583, 20 g barium sulfate (Micropaque), 10 g Dow Corning electronic fluid with viscosity of 20 cs at 25° C, and 8 g Dow Corning catalyst No. 2.

**Methacrylate resins** ZIMMER et coll (1966) evaluated different plastics used in corrosion casts of the central nervous system. The techniques used were similar to those of TOMPSETT (1956). Ethyl and butyl methacrylate modified by other chemicals were blended to make a strong non brittle casting material. Shrinkage studies indicate 6.25 % diminution of volume 24 hours after the material has hardened.

This material is commercially available as Batson's No. 17 corrosion compound (Polysciences Inc., Rydal, Pennsylvania).

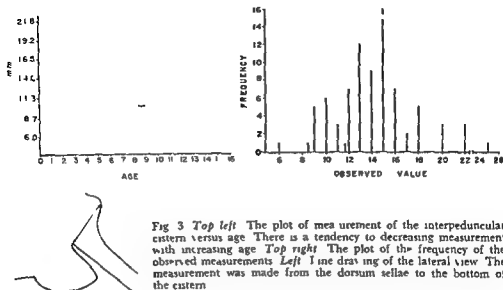


Fig 3 Top left The plot of measurement of the interpeduncular cistern versus age. There is a tendency to decreasing measurement with increasing age. Top right The plot of the frequency of the observed measurements. Left Line drawing of the lateral view. The measurement was made from the dorsum sellae to the bottom of the cistern.

**Polyvinyl acetate** This is simple to use. It requires the addition of acetone and the material is ready for use. The disadvantage is the solubility of acetone in water and formalin. The dissolved polyvinyl does not share this solubility. The cast is distorted as a result. The mixture used exhibited satisfactory hardness without being brittle. Thermolability was another disadvantage.

**Barium gelatin** This mixture is similar to that used by LILJEQUIST. It was prepared well in advance of injection. 50 ml plastic syringes were filled and the material allowed to harden. Immediately prior to use, the syringe was placed under hot tap water. This was sufficient to liquify the gelatin. After several weeks the material was overgrown with bacteria. Antibiotic (Polycillin) was added to the mixture and no growth occurred in storage at room temperature for one year.

The formula used is 50 g barium sulfate (Micropaque) 30 g gelatin 5 g gum arabic 200 ml water and 1 g Polycillin.

The advantages are (1) Low viscosity in liquid phase (2) Liquification in hot water permits preparation of sufficient quantities in advance without the necessity of preparing a fresh batch for each patient (3) Shrinkage does not occur during the first hours.

The disadvantages of this mixture are (1) Solidification is temperature dependent. Hardening may occur before the material is completely injected.

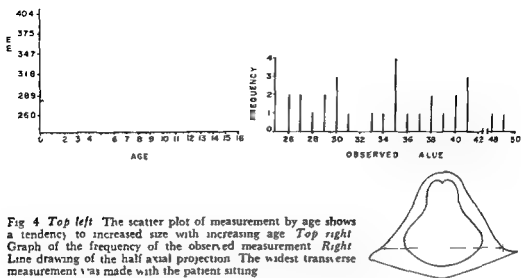


Fig 4 Top left The scatter plot of measurement by age shows a tendency to increased size with increasing age Top right Graph of the frequency of the observed measurement Right Line drawing of the half axial projection The widest transverse measurement was made with the patient sitting

(2) When hardened the barium gelatin is quite brittle. Fragmented pieces fell away from the brain during removal from the calvarium. (3) Within 24 hours some shrinkage was demonstrated.

The corrosion casts made with methyl or butyl methacrylate and vinyl acetate were the most interesting but least feasible for routine casting. Because of the short period of time that the body was available during postmortem examination and the fact that the body was not available for corrosion digestion, these casts were made infrequently. Most of the casting studies were restricted to stillborns and miscarriages.

The material assembled was derived from five hospitals (St. Francis Hospital, Lynwood; White Memorial Medical Center, Los Angeles; Children's Hospital, Los Angeles; Children's Hospital, Orange; Los Angeles County General Hospital Unit II, Los Angeles, Calif.). A search was made for all available encephalographs at these institutions. The films and histories were reviewed to determine if criteria of normality were fulfilled. These cases had the advantage of follow-up study. Those that were subsequently proven to have intracranial disease were eliminated from consideration as were those obvious cases with elevated intracranial pressure or spinal fluid abnormalities.

The encephalographic examinations performed during the course of the project had the advantage of close scrutiny. Care was taken to fill all the cisterns.

The postmortem investigations were made whenever material was available. The results are derived from 91 patients.

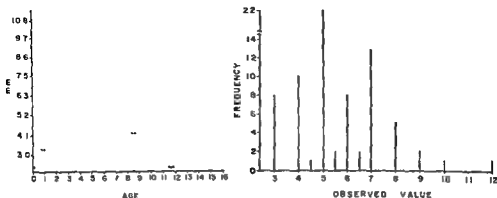


Fig 5 Top left The scatter plot of measurement versus age shows no growth tendency since there are insufficient patients below the age of two Top right Graph of the frequency of observed values Left Line drawing of the lateral view of the pontine cistern measurement The widest measurement was made 1 cm inferior to the posterior clinoid process

## Results

**Interpeduncular cistern** The interpeduncular cistern is measured in the lateral film from the dorsum sellae to the bottom of the cistern. The mean value is 14.1 mm. The standard deviation is 3.50. The values vary from 6.0 to 27.0 mm. This is measured in 82 cases. The 95% confidence limits are 7.10 mm to 21.1 mm. The interpeduncular cistern is larger in children than in adults. The mean value is larger and there is greater variation in size. This corresponds to the larger values in children reported by CARLSSON & LODIN. The plot of age versus measurement shows a negative correlation. The measurements show a general tendency to decrease with increasing age. Perhaps this reflects brain growth (Fig 3).

**Ambient cistern** The ambient cistern is measured in the half axial projection made with the patient sitting. This reflects the problem of incomplete filling in cases from the retrospective review of encephalographies performed in the past. Similarly incomplete filling occurred in some of the cast studies. Overfilling also occurred in the barium cast studies. The mean value is 34.9 mm. Values varied from 26.0 to 49.0 mm. The standard deviation is 6.17. 95% confidence limits are 22.6 to 47.2 mm. Measurements are made in 29 patients. The cistern shows a tendency to increased size with increasing age (Fig 4).

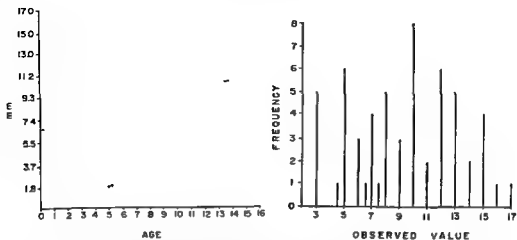


Fig 5 Top left The scatter plot of measurement versus age showed no growth tendency after two years of age. Measurement below the age of two was not sufficient to be conclusive. Top right Graph of the frequency of observed values. Right Line drawing of the lateral view showing the sagittal dimension of the cisterna magna.

**Pontine cistern** The pontine cistern is measured in the lateral film 1 cm inferior to the posterior clinoid process. The mean value is 5.6 mm. The standard deviation is 1.76 mm. The values varied from 3.0 to 12.0 mm. The 95% confidence limits are 2.1 to 9.1. Measurements are from 75 patients. The number of patients under the age of two are too few. After the age of two the cistern does not change significantly in size. The mean values of this series compare with the 5.0 mm value obtained in CARLSSON's series. LILJEQUIST obtained the same value (5.0 mm) in his adult series (Fig 5).

**Cisterna magna** The cisterna magna is measured in the lateral projection. The widest sagittal dimension is measured in films made with the patient sitting. The mean value is 9.25 mm. The standard deviation is 3.76 mm. Values vary from 0.30 to 17.0 mm. The measurement was obtained in 58 cases. The 95% confidence limits are 1.73 mm to 16.7 mm. Great size variability conforms to the size and shape variations observed in adults by LILJEQUIST (Fig 6).

### Acknowledgements

We are indebted to the following physicians for providing us with the additional clinical material that made this investigation possible: John Gwinn, Leo O'Gorman, Merlin Woessner and Marshall Rowan. Special thanks for valuable assistance are extended to Mrs. Marjory Black, Miss Alice Allen, Lawrence Shockey and Mrs. Pat Task. The investigation was supported by U.S.P.H.S. Grant No. NB07394-02, R.M.P. Grant No. 5-G03 RM-00019-02. Statistical calculations were made by Donald Butler.

## SUMMARY

This appraisal of the normal parameters of the cisterns of infants and children is a preliminary investigation. A small number of cases were reviewed. Conclusive statements cannot be made regarding the size, shape and position of the cisterns. However, certain growth trends were noted. Beyond the age of three, the interpeduncular cistern shows a tendency to decrease in size with increasing age; the ambient and pontine cisterns maintain relatively unchanged values after the age of three; the cisterna magna shows great variability in size beyond the age of two. This conforms to the impressions noted in the standard references. This is similar to the findings in adults.

## ZUSAMMENFASSUNG

Diese Wertung der normalen Parameter der Cisternen von Säuglingen und Kindern ist eine Preliminar-Untersuchung. Die Untersuchung ist nur auf eine geringe Anzahl Fälle basiert; eine endgültige Aussage hinsichtlich der Grösse, Form und Lage der Cisternen kann deshalb nicht gemacht werden. Geisse Tendenzen beim Wachstum waren zu beobachten. Nach einem Alter von drei Jahren zeigt die Cisterna interpeduncularis eine Tendenz mit steigendem Alter kleiner zu werden. Die Cisterna ambiens und die Cisterna pontis halten nach einem Alter von drei Jahren relativ unveränderte Werte aufrecht. Die Cisterna magna weist eine grosse Variabilität in ihrer Grösse nach einem Alter von zwei Jahren auf. Das stimmt mit den Eindrücken von den Standardreferenzen überein. Das ist auch den Befunden beim Erwachsenen ähnlich.

## RÉSUMÉ

Cette étude des caractères normaux des citernes du nourrisson et de l'enfant est une étude préliminaire. Les auteurs ont étudié un petit nombre de cas. Ils ne peuvent pas donner des conclusions définitives concernant les dimensions, la forme et la position des citernes. Cependant, ils ont noté certaines tendances au cours de la croissance. Au delà de l'âge de trois ans, la citerne interpedunculaire présente une tendance à diminuer de volume à mesure que l'âge augmente. Les citernes ambiante et prépontique gardent des dimensions relativement fixes après l'âge de trois ans. La grande citerne a des dimensions très variables après l'âge de deux ans. Ceci est conforme avec les impressions notées dans la bibliographie. Ceci est semblable aux résultats chez l'adulte.

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## THE INTRASELLAR SUBARACHNOID RECESS

Some clinical and radiologic observations

by

T. EL GAMMAL and M. B. ALLEN, JR.

Extension of the subarachnoid space into the sella turcica has been reported with increasing frequency in recent years. BULCH (1951) was the first to recognize the condition when he reported on the anatomic examination of 788 sellae seen at post mortem. The presence of such extensions of the subarachnoid space was first reported in the living subject when ROBERTSON (1957) described air extending into the sella at encephalography. A year later ENGELS reported having seen Pantopaque inside the sella turcica during the course of a cervical myelography.

ENGELS also reported a case of intrasellar extension of the subarachnoid recess in conjunction with a parasellar astrocytoma. Another case in which there was herniation of the subarachnoid recess in association with a tumor of the quadrigeminal plate has been described by DU BOULAY (1965). These two are the only cases of subarachnoid recesses extending into the sella in tumor cases we have found in the literature. All other cases of empty sella are reported after surgical or radiation therapy of pituitary tumors.

Until recently only sporadic reports appeared, most of which contained a few cases of the empty sella syndrome. KAUFMAN (1968) reported on eight cases and

Fig 1 A case of calcified ependyoma of the fourth ventricle. During encephalography no air entered the ventricles. Air in the suprasellar region outlines a dilated anterior third ventricle displacing the subarachnoid space inside the sella turcica. Calcification in the posterior fossa.



In 1969 ZATZ et coll reported on 12 such cases. We have recently collected 24 cases in a small Neuroradiological Service at the Medical College of Georgia. Twenty-two of these cases were examined during a period of two years. We believe that the use of tomography and adjustment in the technique of encephalography account for this frequent occurrence.

Empty sella syndrome is the name applied when the subarachnoid space extends into the sella turcica; for this to occur the diaphragm opening must be large. BUSCH has demonstrated considerable variation in the size of this opening.

The dilated third ventricle in cases of hydrocephalus may displace a subarachnoid recess into the sella turcica. Here the arachnoid extension may be secondary to the dilated third ventricle and we have excluded such patients from the present report. An example of such a case is demonstrated (Fig 1).

The purpose of this report is to review the clinical and radiologic features of our 24 patients with subarachnoid recesses extending into the sella. The clinical picture is summarized in three tables. Excluding two patients who had had intrasellar tumors, one in a male and one in a female, the sex incidence was 68% female. Hypertension was noted in 15 patients. CSF proteins were significantly elevated only in one case in a patient with clivus meningioma. Endocrinologic evaluation was not complete; however, the adrenal cortical steroid excretion was reduced in two patients who had had pituitary tumors which had been excised, in two cases of rhinorrhea, and one case with trigeminal neuralgia having marked herniation of the subarachnoid recess into the sella.

Table 1

*Group A Patients with increased intracranial pressure*

Age	Sex	Discharge diagnosis	Head ache	Blood pressure	CSF pressure	CSF cell count	CSF protein	Endocrinologic evaluation
50	♀	Meningioma left sphenoid ridge	No	130/90	150	0	22	Normal adrenal cortical steroid excretion
51	♂	Clivus meningioma	No	120/80	150 (had been 500)	1	80	Not recorded
25	♂	Arteriovenous malformation posterior fossa rhinorrhea*	Yes	145/100	250	1	57.5	Reduced adrenal cortical steroid excretion
26	♂	Benign increased intracranial pressure	No	155/95	300+	0	9	Not done
74	♀	Benign increased intracranial pressure	Yes	110/70	165 (had been 330)	0	18	Only PBI + I
38		Benign increased intracranial pressure*	Yes	200/100	330	20	20	Normal adrenal cortical steroid excretion
48		Rhinorrhea	Yes	180/100	Not recorded	0	30	Slightly reduced adrenal cortical steroid excretion
6	♂	Arrested hydrocephalus	No	90/60	190	2	4	Not done

\* Similar cases were reported by SCHIFFER et coll. and OLIVA et coll.

We categorized our patients into three groups. Group A (Table 1) consists of eight patients with increased intracranial pressure at the time of examination or previously. Included were three patients with intracranial tumors, one case of arrested hydrocephalus, three cases of benign increased intracranial pressure, and one patient with spontaneous rhinorrhea. Group B (Table 2) consists of eight patients with recorded CSF pressures 180 mm of CSF or above, but no other evidence of increased intracranial pressure. There were two cases of trigeminal neuralgia, four cases of vascular disease, one case with seizures, and one case with

Table 2  
Group B Patients with increased cerebrospinal fluid pressure

Age	Sex	Discharge diagnosis	Head ache	Blood pressure	CSF pressure	CSF cell count	CSF protein	Endocrinologic evaluation
50	■	Trigeminal neuralgia	No	160/120	220	0	25	PBI normal Adrenal cortical steroid excretion reduced
38	♀	Trigeminal neuralgia	No	120/75	190	—	—	PBI normal
34	♀	Generalized seizures	Yes	130/70	180	1	7	Normal adrenal cortical steroid excretion
36	♀	Lupus erythematosus	Yes	100/60	230	0	20	Normal adrenal cortical steroid excretion
21	♂	Cephalgia optic atrophy etiology undetermined	Yes	150/105	220 180	1	7	Not done
41	♀	Occlusion of right middle cerebral artery	Yes	120/80 (on anti hypertensives)	240	0	15	Not recorded
55	♀	Arteriosclerosis	Rare frontal	150/85	165 190	2	18	Not done
62	♀	Cerebral thrombosis hypertension	No	260/140	190 210 135	6	15	Diabetes

optic atrophy Group C (Table 3) consists of six cases with normal CSF pressure and two cases with previously resected intracellar tumors, one a craniopharyngioma, and one an eosinophilic adenoma. The latter patient's course had been complicated by intracerebral hematoma following the resection of his tumor. This patient later developed extraventricular hydrocephalus secondary to adhesions at the tentorium. The CSF pressure in this patient was 430 mm of water at the time of his examination.

The findings in conventional films and at encephalography were diagrammed. Group A (Fig. 2) includes four patients with an enlarged sella. Two cases had erosion of the lamina dura. In four cases the depth of the sella was more than the p diameter measured between the dorsum and the tuberculum sellae. The

Table 3

*Group C Patients with normal cerebrospinal fluid pressure and intrasellar tumors*

Age	Sex	Discharge diagnosis	Head ache	Blood pressure	CSF pressure	CSF cell count	CSF protein	Endocrinologic evaluation
33		Hypertension	No	210/130	130	2	10	Not recorded
33	o	Atrophy of right cerebral hemisphere	Past history	125/90	150	0	12	Not done
36	♀	Unconsciousness sella enlarged	Yes	120/70	70	—	—	Normal T4 and T3 Normal adrenal cortical steroid excretion
41	o	Generalized seizures cortical atrophy	No	112/50	130	0	26	Not done
43	o	Temporal lobe seizures	Yes post seizures	135/100	130	1	18	Not recorded
43	o	Recent onset of seizures	Yes chronic	110/70	130	0	14	Not recorded
49	♀	Previous cranio pharyngioma	Yes	120/70	Not measured	1	7	Reduced adrenal cortical steroid excretion
40	o	Previous acidophilic adenoma	Yes	135/80	430	0	50	Reduced adrenal cortical steroid excretion

difference between the two diameters was 4 mm or more. To this type of sella we gave the term deep sella. The anterior end of the third ventricle was low in four and dilated in two. The herniation of the subarachnoid recess was marked in two, moderate in four and mild in two.

Six patients in Group B (Fig. 3) had a large sella. Three cases had erosion of the lamina dura and three cases had a deep sella. The deep sella in Groups A and B constituted 7 of 16 cases. In Group C, however, there was only one case with a deep sella. The anterior end of the third ventricle was low in five and was possibly dilated in one. There was marked herniation of the subarachnoid recess in four and moderate herniation in four cases. In Group C (Fig. 4) the sella was large in five. There was erosion of the lamina dura in one, thinning of the dorsum sellae in one and erosion of the dorsum sellae in another one. One sella was deep. The anterior end of the third ventricle was low in five and dilated in one. Herniation of the subarachnoid recess was marked in one, moderate in two



Fig 2 The findings in conventional films and at encephalography Group A 8 cases with intracranial pressure

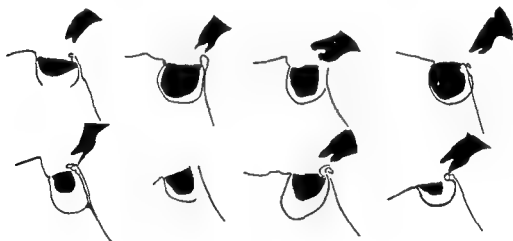


Fig 3 The findings in conventional films and at encephalography Group II 8 cases with high CSF pressure

and slight in four. Of the entire series, fifteen patients had a large sella. In nine patients the sellae had normal size. In five of these, the sella was entirely normal.

Angiography was performed in sixteen patients, a unilateral examination was performed in eight and bilateral angiography in the other eight. Three tumors were found and five cases had vascular disease. Two cases had unilateral small



Fig 4 The findings in conventional films and at encephalography. Group C: 8 cases with normal CSF pressure and intrasellar tumors. \* denotes CSF pressure 430 mm.

aneurysms of the intracavernous part of the carotid arteries and six were normal. Two cases had prominent pterygoid veins near the skull base. One of these had trigeminal neuralgia and increased CSF pressure. The angiogram revealed also a small aneurysm of the intracavernous part of the carotid artery. She had significant herniation of the subarachnoid recess inside a large sella.

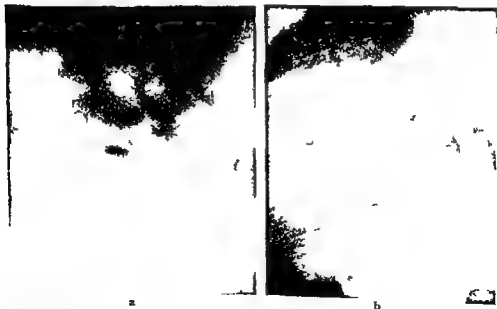


Fig 5 a) Hanging position. Lateral tomography in sitting position. No air is seen inside the sella. b) Hanging position. Lateral tomography in supine position. Air inside the sella.



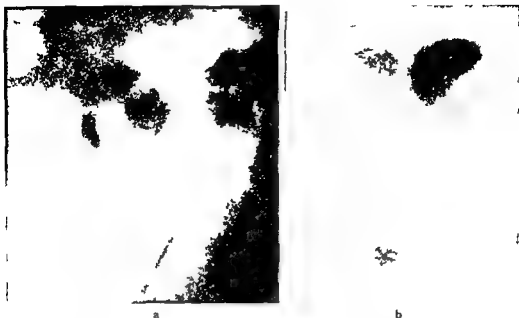


Fig 6 a) Lateral view with tomography. Large intrasellar subarachnoid recess. b) Anterior view.

### Discussion

Encephalography was performed in all twenty-four patients with tomography in twenty-one cases. Films obtained without tomography showing air in the region of the sella may be misleading. In such cases an air-fluid level projected over the sella is confirmatory evidence of an intrasellar subarachnoid recess. Tomography is the technique of choice when examining patients for extensions of the subarachnoid recess into the sella.

In most cases air entered the intrasellar subarachnoid recesses when the head was hyperextended in the sitting position. However, in four cases filling of the recesses occurred only when the head was in the hanging head position with the patient lying supine (Fig 5).

Tomography in the frontal view is usually helpful, especially when there is a large herniation of the subarachnoid space (Fig 6b). The possibility that air in the sphenoid sinus, anterior to the sella, may be projected over the sella region should be borne in mind. In cases with lesser degrees of herniation, tomography may be negative because of inadequate filling of the recess in the supine position. The hanging head position fills the sella subarachnoid recesses best. In this position we cannot obtain a frontal tomogram with our equipment, therefore in our series the lateral tomogram has proved to be more reliable.



Fig 7 Posterior fossa arteriovenous malformation a) Lateral tomogram No air entered the ventricles at encephalography Air is seen in the sella Indentations of enlarged veins into the air filled sellar and suprasellar subarachnoid space b) Left vertebral angiography Lateral view Arteriovenous malformation in the posterior fossa

KAUFMAN et coll (1968) have recommended thumping the occiput in cases where an empty sella syndrome was possible but where air failed to enter this space by simple positioning. This technique was successfully applied in two patients of the series. Air had failed to enter the intrasellar recesses using simple hanging head technique.

The anterior part of the third ventricle was found to lie very close to the dorsum sellae in fourteen of our twenty four patients. ZATZ et coll (1969) demonstrated the frequent association of a low lying third ventricle with intrasellar extension of the subarachnoid space.

From our encephalograms we have concluded that while lateral tomography is indispensable for differentiating enlarged sellae which contain subarachnoid recesses from those sellae filled with neoplastic pituitary lesions, there are still a few cases in which the differentiation is equivocal. Difficulties arise from failure to introduce air into the sella despite proper positioning and manipulation. Whenever the third ventricle is found to be low, every attempt should be made to manipulate air into the sella.

KAUFMAN (1971) reported looping of the proximal part of the anterior cerebral artery in one case of empty sella. This was found in only two cases of this series. We have also seen prominence of the pterygoid veins. Initially we

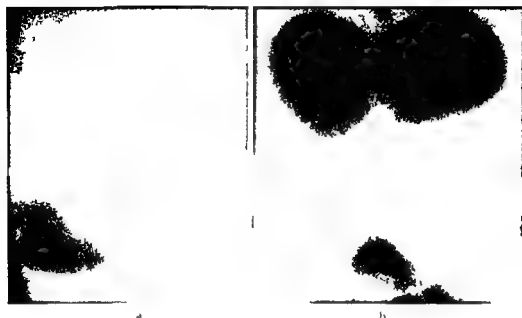


Fig. 8 Left middle fossa meningioma. a) Hanging head. Lateral tomogram in sitting position. Air in the sella turcica. b) Frontal view. The medial edge of the tumor is outlined by air inside and above the sella turcica. Deformity of the left frontal horn.

assumed this to be an indication of increased intracranial pressure, but we have since noted the same in patients who had no evidence of increased intracranial pressure and whose examinations were otherwise normal. These cases indicate that the angiographic findings are not often helpful in identifying patients with intrasellar subarachnoid recesses.

The causes of extension or herniation of the subarachnoid recesses into the sella are probably multiple. Some empty sellae are a result of diminution of the intracellular contents. This process was seen in two of our cases as a result of surgery. Some intrasellar extensions of subarachnoid spaces may occur normally. We agree with KAUFMAN (1968), who believes that many empty sellae are often associated with elevated intracranial pressure. Three of our cases were associated with intracranial tumors.

Four patients in Group A (patients with evidence of increased intracranial pressure at the time of examination or previously) had large sellae turcica. All four of the sellae were characterized by a marked increase in the ratio of depth to a p diameter and were categorized as deep sellae. We had six patients in Group II (patients with measured CSF pressure of 180 mm or above, but no other evidence of increased intracranial pressure) with enlarged sellae. Three of these

were categorized as having deep sellae. Five patients in Group C (normal CSF pressure) had enlarged sellae but only one of these was deep. Thus in the case of a large sella demonstrated on the conventional films of a patient being evaluated for a lesion in the area we favor an empty sella when the pituitary for a demonstrates the characteristics of a deep sella. Confirmation can only be obtained by proper encephalographic examination.

### Conclusion

In conclusion we have found the presence of intrasellar extension of the subarachnoid recesses much more commonly than has been previously reported. Tomography is invaluable in demonstrating this condition. The presence of a deep sella on conventional skull films is suggestive of intrasellar extension of the subarachnoid recesses. However the diagnosis cannot be confirmed without demonstrating air within the sella.

Two of our three patients with intracranial tumors had deep sellae. We have seen deep sellae in association with longstanding benign intracranial tumors and in association with large arteriovenous malformations.

We believe that the deep sella is an indication of elevated intracranial pressure and would like to introduce this type of sella as a new category IV in the classification of DU BOULAY & EL GAMMAL (1966) of sella changes in raised intracranial pressure.

The sella enlargement in some cases of arteriovenous malformation may be the result of intrasellar extension of the subarachnoid spaces rather than carotid enlargement as had been previously suggested REISBACH & POTTS (1966).

### SUMMARY

Extension of the subarachnoid space into the sella turcica has been demonstrated in twenty-four patients. Three of these patients had intracranial tumors. The condition is more common than previously recognized. Tomography and proper positioning of patients are essential in demonstrating its presence. Intracranial hypertension was common among our patients. The term deep sella was introduced. The condition is frequently associated with chronically increased intracranial pressure.

### ZUSAMMENFASSUNG

Die Ausbuchtung des Subarachnoidal Raumes in die Sella turcica wurde bei 24 Patienten nachgewiesen. Drei dieser Patienten hatten intracraniale Tumoren. Diese Bedingungen treten häufiger auf als zuvor erkannt. Tomographie und richtige Lage der Patienten sind wichtig deren Vorhandensein nachweisen zu können. Ein intracranialer Hochdruck war bei unseren Patienten gewöhnlich. Der Begriff tiefe Sella wurde eingeführt. Dieser Zustand ist häufig mit einem chronisch gesteigerten intracranialen Druck verbunden.

## RÉSUMÉ

Les auteurs ont mis en évidence une extension de l'espace sous arachnoïdien dans la selle turcique chez vingt quatre malades. Trois de ces malades avaient une tumeur intracrânienne. L'extension de l'espace sous arachnoïdien dans la selle turcique est plus fréquente qu'on ne pensait auparavant. La tomographie et une bonne mise en place des sujets sont essentielles pour la mettre en évidence. L'hypertension intracrânienne était fréquente parmi leurs malades. Les auteurs proposent le terme de selle profonde. Cet état est souvent associé avec une hypertension intracrânienne chronique.

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## THE UBIQUITOUS 'EMPTY' SELLA TURCICA

by

BENJAMIN KAUFMAN and WILLIAM B. CHAMBERLIN JR.

The empty sella turcica is a term describing the sella turcica when there is a deficient diaphragma sellae and a remodeled pituitary gland so that to gross visual inspection at autopsy or surgery an empty appearance is apparent (Fig. 1) (Busch 1951; KAUFMAN 1968). Ubiquitous is used to emphasize the prevalence of the empty sella turcica. Radiographically the term empty sella is being used when air or positive contrast is seen below the postulated but not proven position of the diaphragma sellae. The following autopsy investigation was done to evaluate the gross appearance and incidence of the intrasellar subarachnoid space.

### Materials and Methods

En bloc autopsy specimens of the were obtained during the period 1961-1968. Patients referred from other institutions could not be recovered within a selected group, but no patient in the age group ranged from 30 to 93 years with pathographed with a ruler (Figs. 1-2).

sella turcica from 89 consecutive autopsies of 96 patients at Highland View Hospital and have chronic illnesses which are listed. As such they constitute a highly selected group had endocrine disease. Their mean age of 66.5 years. Specimens were fixed in 10% neutral formalin and

were decalcified before staining with hematoxylin and eosin. Roentgenograms were taken before decalcification in three standard projections (anterior posterior, submentovertical, and lateral).

BUSCH's criteria of the extent of diaphragma sellae deficiency and the state of the pituitary gland were applied (BUSCH 1951). His criteria, based on visual and histologic examinations, are:

Type 1 A. The diaphragma sellae forms a complete covering.

Type 1 B. A slight funnel-shaped depression is present in the intact diaphragma sellae.

Type 2 A. A 3 mm or less opening in the diaphragma sellae exists around the hypophyseal stalk.

Type 2 B. A slight funnel-shaped indentation toward the middle of the diaphragma sellae is present.

Type 3 A. The diaphragma sellae is a peripheral rim of tissue 2 mm or less in extent with the pituitary gland freely exposed and covered with arachnoid.

Type 3 B. The diaphragma sellae deficiency is the same as Type 3 A, but the pituitary gland is indented, often eccentrically.

Type 3 C. In addition to the deficient diaphragma sellae characteristic of Type 3, the remodeling of the pituitary gland is marked and the gland may not be apparent on visual inspection. Hence the term "empty sella." Histologically the pituitary gland is markedly remodeled (BUSCH, KAUFMAN 1968). No cellular abnormalities are distinguishable in the remodeled pituitary gland.

However, for the purposes of this investigation, visual inspection only of the autopsy specimens formed the basis for the initial division into the three main types and subgroups of BUSCH. The 89 cases were classified as follows on the basis of the intactness of the diaphragma sellae and the form of the pituitary gland:

Type	No. of cases
1	25
2 A	3
2 B	9
3 A	12
3 B	19
3 C	21

For example, if the sella turcica was deep but the diaphragma sellae was intact and not depressed, the specimen was considered as Type 1 A, and no attempt was made to relate to the volume of the sella turcica. Sagittal histologic sections of the 21 specimens which visually were considered Type 3 C were obtained to evaluate the form of the pituitary gland (Figs 1, 2).

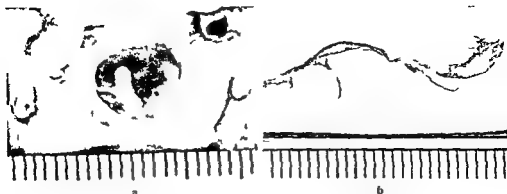


Fig 1 A 36 year old female a) The classical appearance of an empty sella turcica with an almost totally deficient diaphragma sellae and an invisible pituitary gland on inspection Scale 1 div = 1 mm b) Sagittal section of (a) showing remodeled anterior pituitary gland to measure less than 1 mm in its most anterior portion An artefact of preparation has resulted in loss of dorsum and floor This specimen was considered to be Type 3C of BUSCH While the sella was not roentgenologically enlarged the intrasellar subarachnoid space could be relatively appreciable at encephalography

## Results

Applying the visual criteria of a deficient diaphragma sellae and a non visible pituitary gland showed that 21 out of the 89 specimens (23.5%) had the gross appearance of BUSCH's Type 3C the empty sella. Sagittal histologic sections of the 21 sellas showed that only six of the 21 (28.5%) truly had a remodeled pituitary gland to the extent described by BUSCH. Thus only six specimens of the total 89 (6.7%) were finally considered to be Type 3C (Fig 1). In 14 of the 21 specimens (visual Type 3C) the pituitary gland was only moderately flattened and satisfied the criteria of Type 3B (Fig 2). The remaining one had multiple arachnoid strands which has been called a fenestrated diaphragma sellae (McLACHLAN et coll 1968 p 782) and which would not act as an efficient barrier to cerebrospinal fluid (Fig 3) and was considered as Type 3B of BUSCH.

Visual Types 2 and 3 those with deficient diaphragma sellae constituted 71.9% (64/89) of the series.

## Discussion

One meaning of ubiquitous is capable of occurring or appearing anywhere or in many places (WEBSTER 1959 p 2478). We feel this word can be used to describe the occurrence of empty sellas. Autopsy series have established the





Fig 1 A 72 year old female a) An empty sella to visual inspection Scale 1 div = 1 mm b) The histologic sagittal section shows no appreciable flattening of the gland but there is a definite intrasellar subarachnoid space This specimen was considered finally Type 3 II of BUSCH rather than Type 3 C Longest sagittal dimension is 16 mm Scale 1 div = 1 mm Two features illustrated are 1 the deficiency of the diaphragma sellae is no indication as to the size of the sella turcica and 2 with magnification on a roentgenogram and the thin dorsum the question of enlargement secondary to an intrasellar neoplasm could be raised

incidence of types of incompleteness of the diaphragma sellae and essentially our series does not differ (SCHAEFFER 1924 BUSCH, BERGLAND et coll 1968) The work of BUSCH has given us a starting figure of 5.5% incidence of empty sellas in 788 autopsies consisting of two groups of specimens Detailed measurements of 342 sellas were made and 18 of the empty type were present He showed that empty sellas as a group were larger than the other sellas in this group In the combined group of 788 autopsies empty sellas were more frequent in females (34 females 6 males) In our series, 21 of the 89 specimens had the appearance of an empty sella (Figs 1 2 3) The significance of this is that on roentgenograms contrast material would be below the line joining the tuberculum sellae to the top of the dorsum sellae the postulated position of the diaphragma sellae (ENGLES 1958 DI CHIRO 1961 TAVERAS & WOOD 1964) As pointed out by McLACHLAN et coll (1968) it may be difficult to define the tuberculum sellae the point of anterior attachment to the diaphragma sellae In some sellas the anterior wall may be quite long leading to a fictitious appearance of deepening of the sella and the impression of increased volume He also illustrated downward variations in the diaphragma sellae all below the position of the diaphragma sellae if one uses the plane from the tuberculum sellae to the tip of the dorsum as representative of the diaphragma sellae The three forms

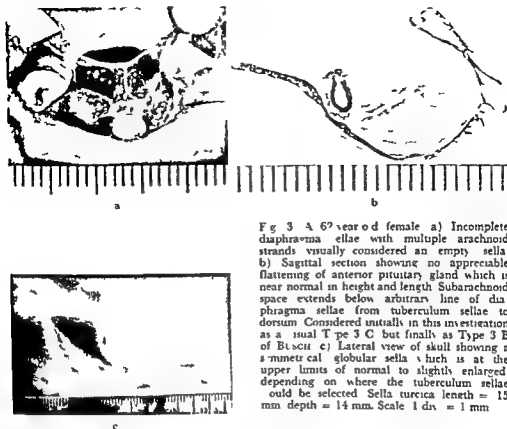


Fig 3 A 67 year old female a) Incomplete diaphragma sellae with multiple arachnoid strands visually considered an empty sella b) Sagittal section showing no appreciable flattening of anterior pituitary gland which is near normal in height and length Subarachnoid space extends below arbitrary line of diaphragma sellae from tuberculum sellae to dorsum Considered initially in this investigation as a usual Type 3 C but finally as Type 3 B of BUSCH c) Lateral view of skull showing a symmetrical globular sella which is at the upper limits of normal to slightly enlarged depending on where the tuberculum sellae could be selected Sella turcica length = 15 mm depth = 14 mm Scale 1 div = 1 mm

of BUSCH's Types 1 B and 2 B Air or Pantopaque in the c patients in the region of the sella would have given a fictitious appearance of an empty sella

Since the diaphragma sellae may be concave or may be partially or totally absent it probably would be more accurate to refer to an intrasellar subarachnoid space as exemplified by demonstration of the intra sellar air or Pantopaque at contrast examinations (ENGLES FERNER 1960 TAVERAS & WOOD 1960 NADJMI 1965 KAUFMAN 1968 McLACHLAN et coll 1968 ZATZ et coll 1969)

What is being seen and reported with increased frequency in the literature is demonstration of intra sellar air with the assumption not necessarily correct that the radiographic sella turcica reflects the position of the diaphragma sellae and the size and shape of the pituitary gland DI CHIRO in 1960 and DI CHIRO & NELSON in 1962 referring to their own cases and the work of BUSCH clearly point out the discrepancy which may exist between a large but empty sella and the pituitary gland which is not enlarged We would like to emphasize that the



Fig 4 A 34 year old male a) Lateral brow up transverse view of sella turcica during cephalography in a male with a history of vertigo and headaches but without neurologic or endocrinologic findings. Anterior extension of sella turcica but no involvement of chiasmatic sulcus or elevation of planum sphenoidale. The dorsum is thin but not displaced posteriorly. Air fluid level (→). This case illustrates asymmetrical enlargement of anterior portion of sella turcica. Scale 1 div = 1 mm b) The sella appears completely air filled. No surgical or autopsy proof of this case. It is presumed that the pituitary gland is markedly remodeled in the bottom of the sella. Anterior wall of sella turcica remodeled. (Case kindly donated by Dr George Wortzman of the Toronto General Hospital Toronto Canada.)

demonstration of a large intrasellar subarachnoid space with contrast material does not in itself prove a flattened or remodeled pituitary gland (Figs 2, 3). As long as the sella is not enlarged too much and there are no endocrine symptoms, clinical problems are rather minimum. However, when there is non tumorous enlargement of the sella with or without endocrinopathy, then the problem of etiology and treatment becomes important (KAUFMAN, 1968). Empty sellas are part of the everyday medical practice and are not rare or mysterious. Non tumorous enlargement of the sella turcica, symmetrical or asymmetrical, is only part of a spectrum and as such will have a lower incidence than the overall group of empty sellas. The terms intrasellar air, remodeled pituitary gland, and non tumorous enlargement, whether symmetrical or asymmetrical, are probably preferable to the term empty sella.

If the concept is accepted that a deficient diaphragma sellae of a few millimeters is sufficient to allow cerebrospinal fluid to enter the hypophyseal fossa and enlarge this part of the sella turcica, then 71.9% (64/89) of the Highland



Fig 5 A 38-year old female: a) Brovi up transverse view during encephalography in an obese female with amenorrhea and hypothyroidism. Air has not entered the 19 mm  $\times$  19 mm sella and an intrasellar neoplasm cannot be excluded. b) Following percussion of head in supine head hanging position, air is seen within the sella. Percussion can be employed either in the erect position or in the recumbent position providing that air is in contact with the sella turcica as seen in (a). c) A lateral tomogram confirms the intrasellar position of the air and excludes a neoplasm as the cause of the globularly enlarged sella turcica. Subsequent examinations failed to prove hypopituitarism and the patient is considered as having a non tumorous enlargement of the sella turcica (empty sella) exhibiting no endocrine or sella turcica changes during a three year follow up period. Scale 1 div. = 1 mm.

View series would have been capable of exhibiting intrasellar subarachnoid contrast material or sellar enlargement at some time under the proper circumstances (ENGLES, FERNER, DI CHIRO 1961, TAVERAS & WOOD, DU BOULAY 1965, VON NADJMI, DU BOLLAY & EL GAMMAL 1966, KAUFMAN 1968, ZATZ et coll.). The microscopic arrangements of the meninges in the hypophyseal fossa and the resultant cerebrospinal fluid spaces have been described (ENGLES, FERNER, DU BOLLAY & EL GAMMAL) but their role in the development of non tumorous enlargement of the sella turcica or normal sized empty sellas is not clearly defined. An incomplete diaphragma sellae apparently is the prime requisite (KAUFMAN 1968, MORTARA & NORRELL 1970) and the dynamic forces of the pulsating cerebrospinal fluid probably cause the remodeling of the bony sella turcica and pituitary gland (FERNER, DU BOULAY 1966, DU BOULAY & EL GAMMAL, KAUFMAN 1968). If non tumorous enlargement is so widespread and not infrequent the question arises why it has not been appreciated before. Perhaps the lesson learned from that famous Scandinavian fairy tale by Hans Christian Andersen, 'The Emperor's Clothes' is most appropriate. Empty sellas have always been present and do not represent a new occurrence (TWYNING 1939, ENGLES, DI CHIRO 1961, DI CHIRO 1961, TAVERAS & WOOD). Probably the first radiographically and surgically proven case of an empty sella was

by TWING in 1939 (his Fig 14 b) The inability to assign numerical values to an individual sella turcica and late that its larger size is secondary to a tumor has been appreciated by many (DI CHIRO & NELSON TAVERAS & WOOD, DU BOULAY & EL GAMMAL, LOMBARDI 1967) (Figs 2, 3) Documented progressive asymmetrical enlargement without eye signs has always been taken to mean an enlarging tumor confined by an intact diaphragma sellae or that the tumor does not touch the optic apparatus In the e cases, radiation therapy was considered the treatment of choice and since non tumorous enlargements do not progress to give definitive and clear cut symptoms of endocrinopathy and eye findings were absent unless herniation of the optic nerves occurred (LEE & ADAMS 1968 MORTARA & NORRELL), the results were considered to be excellent (KAUFMAN 1968) Failure to identify a tumor at surgery in cases of empty sella was attributed to shrinkage of the tumor, migration of the tumor to the sphenoid sinus or simply inadequate surgical exposure (personal communication with neurosurgeons) Fig 4 illustrates asymmetrical enlargement of the anterior portion of the sella turcica in a male without eye findings and with the clinical history of vertigo and headaches Endocrinopathy was not present Progressive asymmetrical enlargement in autopsy and encephalographically proven cases of empty sella occur as part of the spectrum of non tumorous enlargement and is not infrequent (unpublished data KAUFMAN & CHAMBERLIN) To date no congenital empty sella have been seen, suggesting that the remodeling of the pituitary gland and sella turcica is an acquired process dependent upon the intactness of the diaphragma sellae and the cerebrospinal fluid pulsations and pressures (BUSCH FERNER DU BOULAY & EL GAMMAL KAUFMAN 1968) It is well documented that when the diaphragma sellae is destroyed by tumor or removed surgically a mechanical arrangement exists resulting in some cases in progressive non tumorous enlargement of the sella turcica the so-called surgical empty sella (COLBY & KEARNS 1962, ROBERTSON 1967 LEE & ADAMS, MORTARA & NORRELL)

Angiography has been misleading and the more recent trend to utilize angiography and omit encephalography when a pituitary tumor is suspected and radiation therapy is the treatment contemplated has delayed identification in many cases until autopsy is done The enlargement of the sella turcica caused by the intrasellar position of the cerebrospinal fluid also causes lateral displacement of the non bony walls of the hypophyseal fossa causing a lateral displacement of the cavernous sinus and the carotid artery (KAUFMAN 1970) The extent of displacement may exceed the numerical value given by BILL & SCHUNK in 1962 and the qualitative relationships of the normal, more medial position of the posterior part of the intercavernous portion of the internal carotid artery relative to the anterior part may be reversed (CILASE & TAVERAS



Fig 6 A 70-year-old male. Asymmetrically remodeled pituitary gland with a deficient diaphragma sellae. The pituitary gland is displaced posteriorly and to the right. The floor of the sella turcica could be seen anteriorly and to the left. The conventional films of the skull showed a normal small sella turcica. Routine autopsy finding in a patient with no endocrine problems (Not part of the 89 consecutive autopsies.)

1961). It should be stressed that the criteria given by BULL and SCHUNK and by CHASE and TAVERAS are accurate and valuable and do reflect intrasellar masses but one cannot distinguish the contents of the sella with angiography. On lateral arteriograms the carotid siphon may appear closed or open depending on whether the bulging lateral walls of the hypophyseal fossa are primarily below or above the horizontal part of the internal carotid artery. Cavernous sinus angiography reflects the position of the venous sinus but does not indicate the contents of the sella turcica. None of the empty sellas cause an elevation of the anterior cerebral arteries. Herniation of the anterior cerebral arteries into the sella may occur (KAUFMAN 1970 MORTARA & NORRELL).

Encephalography after radiation treatment showing intrasellar air, has been misleading when the assumption is made that the pituitary gland shrunk secondary to the therapy (KAUFMAN 1968). Encephalography remains as the definitive pre mortem diagnostic test. Filling the hypophyseal fossa may easily occur but occasionally difficulties are encountered. Then with the head extended either sitting or recumbent so that the air is in contact with the upper part of the sella a firm tapping or percussion of the head may be needed to dislodge the fluid so that the air may enter the fossa. Fig 5 illustrates the sequence of events in a patient who was subsequently proven to have no evidence of an endocrine deficiency or pituitary neoplasm. An air soft tissue interface with the convexity upward is strongly in favor of an intrasellar mass. Many times the interface is concave downward and yet the contents of the hypophyseal fossa are greater than the expected value of a normal pituitary gland (RASMUSSEN 1924 DI CHIRO DI CHIRO & NELSON McLACHLAN et coll 1968). Some of these cases may contain air beneath the diaphragm and as such the air fluid interface does not represent the upper surface of the gland (McLACHLAN et coll 1968). The

growth pattern of a pituitary neoplasm results in a convex upper surface rather than concave. Practically, if no clinical symptoms or eye findings are present in these cases of a concave diaphragma sellae and a volume exceeding that of a normal pituitary gland, therapy is withheld until the situation is clearly defined clinically. A fenestrated diaphragma sellae or multiple arachnoid strands (Fig 3) or one with a small hole may entrap the cerebrospinal fluid during encephalography, necessitating the tapping maneuver. The pituitary gland may be flattened symmetrically, resembling a bowl displaced posteriorly, and flattened asymmetrically (Fig 6) or flattening of the anterior part with no appreciable gross change in the posterior pituitary may occur (KAUFMAN 1968). Asymmetrical flattening of the pituitary gland can be associated with roentgenologically asymmetrical enlargement of the sella turcica or with no sella change.

The frequency of empty sellas, enlarged and non enlarged, is great enough in the general adult population, male and female, so that they can be found in association or concomitantly with many disease states including the endocrinopathies. An example, the Forbes-Albright syndrome is galactorrhea, amenorrhea, and low urinary follicle stimulating hormone with an enlarged sella turcica associated with a pituitary neoplasm (FORBES et coll 1954). Surgical proof of chromophobe adenomas in some of these patients is the basis for the syndrome. However, we have an autopsy proven case of a Forbes-Albright syndrome treated 15 years prior to autopsy that had an asymmetrically enlarged sella turcica grossly radiographically, and histologically, an eccentrically flattened pituitary gland which was normal without evidence of neoplasm or fibrosis. Another case of Forbes-Albright is available to us in which the encephalogram showed air within the sella and a small remodeled pituitary gland (unpublished data KAUFMAN & PEARSON).

The time has come to demand tissue diagnoses, surgery, or autopsy, in those cases with endocrinopathies and large sellae, either symmetrically or asymmetrically enlarged, which still satisfy the conventional film findings associated with non tumorous enlargement of the sella before assigning these cases to a definitive group. Classically, empty sellas do not cause any alteration of the anterior clinoids or change in the planum sphenoidale. Bone destruction is not a feature and remodeling takes place over many years. A sloping floor may appear to be destroyed, but careful radiographic evaluation will show that destruction is not present. The dorsum may be thinned and bowed slightly posteriorly (Figs 2-3) but is not displaced vertically, as is true with some intrasellar chromophobes (TAVERAS & WOOD DU BOULAY 1965, ROSS & GREITZ 1966, LOMBARDI, KAUFMAN 1968). The configuration of the sella may be globular and symmetrical, asymmetrically enlarged in any direction, or may be one of the so-called deepened sellas (KAUFMAN 1968) in which the dorsum is in line with

the clivus, the anterior wall of the sella is continuous with the chiasmatic sulcus so that at the anterior wall forms a straight line going from the planum down to the depths of the posterior inferiorly increased dimension of the sella. Encephalography is the definitive diagnostic test. But to firmly establish the role of empty sellas in endocrine diseases if any will require histologic and physiologic proof. Well documented cases of symptoms related to empty sellas are present in the papers of DI CHIRO et coll 1968, BRISMAN et coll 1969 and KAUFMAN 1969 referring to cerebrospinal fluid leaks. Eye findings relative to herniations of the optic chiasm and optic nerves into the sella have been described by LEE and ADAMS in 1968, MORTARA and NORRELL in 1970, and GOLBY and HEARNS who gave the original description in 1962. Care should be taken at this early stage in the history of the empty sella complex in attributing too many endocrine abnormalities to it (CAPLAN & DOBBS 1969). Our current approach is that empty sellas are ubiquitous and common enough that they may and do occur concomitantly with many disease states as well as in individuals who are considered normal by all existing medical standards.

At autopsy, one should not depend on the size of the deficient diaphragma sellae to estimate the size of the sella turcica (Fig 2). From these autopsy specimens it is obvious that the size of the diaphragma sella opening does not reflect the extent or configurations of the enlarged bony margins and fibrous walls of the sella turcica. It is necessary to section the sella or inspect beneath the diaphragma sellae.

### Acknowledgements

The authors wish to express their gratitude to Olof H. Pearson, Professor of Medicine, Section of Endocrinology, Case Western Reserve University for his valuable assistance in endocrinologic matters and to Barbara H. Kaufman for her efforts in the preparation of this paper. This work was supported in part by RR 80-08 of the National Institutes of Health Clinical Research Center Grant.

### SUMMARY

Specimens of the sella turcica from 89 consecutive autopsies formed the basis for evaluation of the incidence of incompleteness of the diaphragma sellae and occurrence of empty sella turcicae. The common occurrence of an intrasellar subarachnoid space is emphasized. The effect of the intrasellar subarachnoid space on the bony sella and non bony lateral walls of the sella is related to conventional film and angiographic findings. An encephalographic maneuver to enhance air filling of the intrasellar subarachnoid space is described.

### ZUSAMMENFASSUNG

Proben der Sella turcica von 89 nacheinanderfolgenden Autopsien bilden die Basis um das Vorkommen unvollständiger Diaphragmas der Sella und das Vorhandensein



einer leeren Sella turcica festzustellen. Das normalerweise Auftreten eines intrasellaren Subarachnoidalraums wird hervorgehoben. Der Einfluss des intrasellaren Subarachnoidalraums auf die knöcherne Sella und die nicht knöchernen Seitenwände der Sella wird zu konventionellen Film- und angiographischen Befunden in Beziehung gestellt. Es wird ein encephalographisches Verfahren um die Luftfüllung des intrasellaren Subarachnoidalraums zu erhöhen beschrieben.

## RÉSUMÉ

Les selles turciques prélevées au cours de 89 autopsies consécutives ont servi à évaluer la fréquence des diaphragmes sellaires incomplets et des selles turciques vides. Il existe souvent un espace sous arachnoïdien intrasellaire. Les auteurs établissent une relation entre l'effet de l'espace sous arachnoïdien intrasellaire sur la selle osseuse et sur les parois latérales non osseuses de la selle et les résultats de la radiographie simple et de l'angiographie. Ils décrivent une manœuvre encephalographique qui facilite la pénétration de l'air dans l'espace sous arachnoïdien intrasellaire.

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## THE SHAPE OF THE LATERAL VENTRICLES IN CEREBRO-VASCULAR DISEASES

by

PIOTR KOZŁOWSKI and JERZY DĄBECKI

Little experience is available as to the interpretation of encephalographic appearances characteristic of vascular lesions of the brain. Encephalography is carried out in cerebro-vascular diseases only occasionally (and then usually in cases of probable cerebral tumour). The examination will often disclose dilatation of one of the lateral ventricles and sometimes irregularities of the outlines of the walls (Fig. 1). The present investigation constituted an attempt to establish a morphologic basis for these findings as well as to determine their relationship to the character of vascular lesions of the brain. Post mortem encephalography as well as macro- and microscopic examinations of the brain were performed in 50 cases with neurologic signs and symptoms of cerebro-vascular disturbances.

The ventricular system failed to fill in 8 cases in which autopsy revealed damage of one hemisphere by hemorrhage and cerebral oedema. The ventricular system was filled with blood in 5 cases and unsatisfactorily filled in 12 cases, brain infarction with severe cerebral oedema was evident at autopsy.

Dilatation of one or both ventricles was present in 24 cases. Encephalography disclosed asymmetric dilatation of the ventricular system in 15 cases of this group (Fig. 2). Specimens of the brain revealed focal tissue necroses within either the

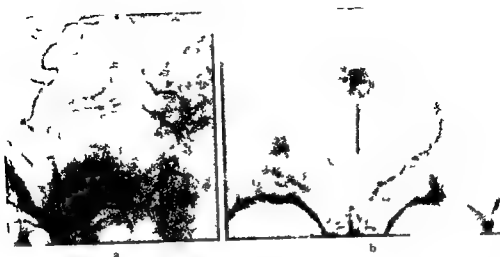


Fig 1 Male aged 45 with aphasia and right hemiparesis a) Left lateral aortic angiogram occlusion of the anterior group of candelabra branches b) Aortic angiogram Dilated lateral ventricles the left more than the right irregular outline of the lateral wall of the left ventricle Enlarged third ventricle slightly to the left of the median plane Widening of subarachnoid space of left hemisphere

cortex or the white matter (Fig 3) Irregular outlines of the lateral walls of the lateral ventricles were observed in the remaining 9 cases (Fig 4) Coronal sections of the brains demonstrated small subependymal cavities or gliomeo dermal cicatrices shrinking the ventricular walls and subependymal structures (Fig 5) No changes were evident in 6 cases

### Discussion

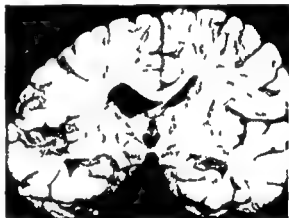
Many conditions may produce widening of the ventricular system of the brain (HOLMGREN 1947 BRUIJN 1959 LÖNNVU 1966 KOZŁOWSKI 1967) The outlines of the lateral ventricles in aortic angiograms are commonly smooth and clearly visible Dilatation of a lateral ventricle is however sometimes accompanied by irregular outlines of its walls According to BERGERON (1967) the following conditions may produce irregularities of the ventricular walls of the brain tuberculous sclerosis choroid plexus papilloma, ependyma seeding from malignant tumours granular ependymitis and ribbing of the ventricular wall in hydrocephalus

The irregularities may also be caused by periventricular arteriovenous malformations protruding into the lumen of the lateral ventricles (LINDGREN 1954) The present material suggests that in addition irregularities of the lateral walls of the lateral ventricles produced by foci of vasculogenesis may occur in the

Fig. 2 Male age 70 with left hemiparesis and general atherosclerosis. Ap. encephalogram. Enlargement of the right lateral ventricle with ipsilateral widening of subarachnoid space.



Fig. 3 Same case as in fig. 2. Coronal section of brain. Encephalomalacia with cicatrization affecting almost entire superior gyrus of the right temporal lobe. Widening of right Sylvian fissure.



basal ganglia of the brain. Multiple small cavities or glione odermal cysts sometimes appeared in the subependymal area of the caudate nucleus; these cause shrinkage of the subependymal glial layer and ependyma with resulting deformation of the ventricular walls. Localisation of the vasculogenesis within the basal ganglia could be explained by the characteristic predilection of this region to arteriolo sclerosis (MARKIEWICZ 1966, DYMICKI 1968).

Symmetric or asymmetric dilatation of the ventricular system with irregular outline of the inferolateral part of the lateral ventricular wall is often observed in cerebrovascular disease; changes of this kind may be bilateral, when the vascular lesions are distant from the ventricular system, e.g. in the cortex or white matter, the ventricular wall is smooth and the ipsilateral subarachnoid



Fig 4 Female aged 63 Ap encephalogram Cerebral atherosclerosis and extrapyramidal syndrome with dilatation of lateral ventricles Irregular and deformed outlines of the lateral wall of both ventricles

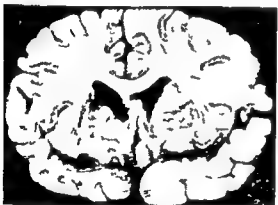


Fig 5 Same case as in fig 4 Coronal section of the brain Small subependymal gliomas in the right caudate nucleus shrinking the ventricular wall and subependymal structures Other sections revealed small cicatrices in both caudate nuclei with shrinking of lateral walls of the ventricles

space often increased Any enlargement of the ventricular system accompanied by irregular outline of its lateral walls is however most probably due to gliomas or cicatrices in subependymal structures of the brain

## SUMMARY

Encephalography with macro and microangiography in 50 cases with neurologic symptoms and asymmetry of the lateral ventricles were studied. Irregularity of the ventricular walls may be due to shrinking of subependymal structures.

Examinations of the brain were performed in 50 cases with neurologic symptoms and asymmetry of the lateral ventricles. It is suggested that the irregular outlines of the lateral ventricles are due to shrinking of subependymal gliomas or cicatrices.

## ZUSAMMENFASSUNG

Eine Encephalographie mit makro und mikroskopischen Untersuchungen des Gehirns wurde an 50 Fällen mit neurologischen Zeichen einer cerebrovasculären Erkrankung post mortem vorgenommen. Dilatation und Asymmetrie der lateralen Ventrikel waren gewöhnlich vorhanden. Es wird vermutet, dass die Unregelmässigkeit der Ventrikelwände auf subependymalen gliomesodermalen Narben, die die Strukturen schrumpfen lassen, beruht.

## RÉSUMÉ

Les auteurs ont pratiqué des encephalographies et des examens post mortem macro et microscopiques du cerveau sur 50 sujets qui presentaient des signes radiologiques d'affection cerebro vasculaire. Il y avait dans la plupart des cas une dilatation et une asymétrie des ventricules lateraux. Les auteurs pensent que l'irregularité des parois ventriculaires peut être due à des cicatrices gliomesodermiques sous endymaires qui retractent les structures sous endymaires.

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## PONTINE ANGLE TUMOURS

### Differentiation between extra and intra axial tumours

by

L O LÄNNER D LINDELL and M ROSENCRANTZ

With the aid of encephalography it is as a rule possible provided a correct technique is used to diagnose even small tumours in the pontine angle and also to differentiate between intra and extra axial tumours. The differential diagnosis is mainly based on the appearance of the cisterns. An extra axial process usually bulges into the pontocerebellar and medullary cisterns from the lateral side whereas an intra axial process compresses them from above and from the medial side. A small acoustic neurinoma may be almost completely outlined by gas in the pontocerebellar cistern. If the tumour is larger it displaces the brain stem towards the opposite side. Because of that the cisterns of the tumour side become widened whereas the corresponding cisterns of the opposite side are compressed. The displacement of the brain stem also results in a typical displacement and rotation of the fourth ventricle. The rotation may give an impression of a distortion of the fourth ventricle, which may lead to a false diagnosis of an intra axial tumour. In some cases however, an extra axial tumour bulges into the brain stem instead of displacing it. An asymmetrically located pontine tumour or a tumour of the fourth ventricle growing through the foramen of Luschka may on the other hand extend into and more or less completely compress the cistern, which accordingly cannot be gas-filled at the examination. In these cases it is sometimes difficult even at operation to find out whether the tumour is



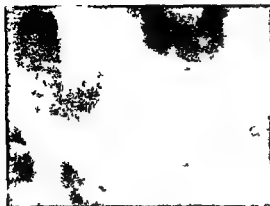


Fig 1



Fig 2

Fig 1 Tomography. Medium sized acoustic neuroma on the left side surrounded by cisternal gas

Fig 2 Large acoustic neuroma. Tumour pole (→) in widened medullary cistern. Compression of contralateral cisterns and rotation of the 4th ventricle

extra or intra axial. For the differential diagnosis in these cases vertebral angiography may be of value. The angiographic diagnosis is based partly on vascular displacement and partly on demonstration of tumour vessels. Tumour vessels in the form of a net of very fine vessels have been described in acoustic neuroma by some authors (OLSSON 1953, GOREE *et coll.* 1964, PEETERS 1969) but are in our experience only rarely seen. Displacement of arterial branches are in our opinion often difficult to evaluate for one thing because of the many normal variations. Displacement of veins as described especially by HUANG *et coll.* (1968) seem to be more reliable.

Even if it is thus in most cases possible to distinguish between extra and intra axial tumours we have sometimes made mistakes in both directions i.e. diagnosing extra axial tumours as possibly intra axial and vice versa. In order to find out the reason for this and if possible improve our accuracy in this respect we have reviewed our cases of tumours in the pontine angle area.

**Material.** The review covers the years 1953–1969. Unfortunately the quality of the examinations during this period varies considerably. This is partially due to the technical equipment. A film changer for complete serial angiography and equipment for tomography were not available before 1959. Furthermore the condition of some patients was too poor to allow a complete neuroradiologic investigation. Of the 150 cases reviewed 126 were extra axial (112 acoustic neuromas, 10 meningiomas, 1 neuroma of the 6th nerve, 1 of the 12th nerve, 1 cholesteatoma and 1 arachnoidal cyst) and 22 were intra axial (13 pontine tumours, 5 fourth ventricle tumours, 3 plexus papillomas and 1 tumour of the

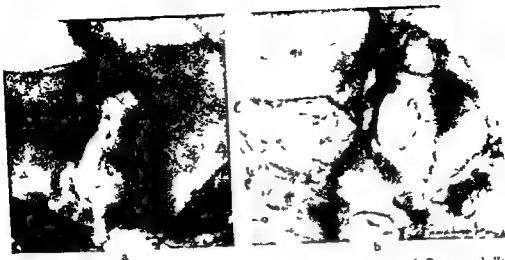


Fig 3 Left side meningioma originating in close relation to the porus a) Pontocerebellar cistern compressed from the medial side b) Petrosal vein and tributaries elevated

cerebellar hemisphere) In 2 cases exploration revealed no tumour Encephalography was performed in practically all cases even in those with signs of increased intracranial pressure Ventriculography was performed in those cases in which no gas entered the ventricular system and a definite diagnosis could not be made from the appearance of the cisterns Vertebral angiography was carried out in only about 1/3 of the cases This examination was in some cases somewhat incomplete partly because of inadequate equipment and partly because the contrast material in some cases was injected in the opposite vertebral artery resulting in an incomplete filling of the vessels of the tumour side

In the majority of cases characteristic changes were demonstrated at encephalography making a correct preoperative diagnosis possible (Figs 1 2) In 11 cases however the preoperative diagnosis was incorrect regarding extra- or intra-axial location Thus in 4 cases an intra-axial tumour was diagnosed as probably extra-axial while 4 extra-axial tumours were interpreted as being most likely intra-axial In 2 cases diagnosed as extra-axial masses no tumour was found at operation

When reviewing the films of these 10 cases we feel that a correct diagnosis should have been possible in 7 of them Two of these 7 tumours were meningiomas In one case the pontocerebellar cistern was compressed from the medial side which was taken as evidence of an intra-axial tumour (Fig 3 a) The explanation for this non-characteristic appearance is apparently that the cistern from above the cistern being gas-filled only the fourth ventricle was however displaced and rotated



Fig 4 Subtraction film. Large left side acoustic neurinoma displacing the petrosal vein (→) medially and upwards. Right petrosal vein (←) in normal position.

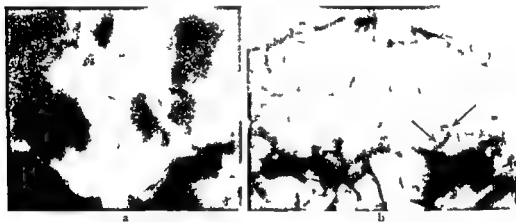


Fig 5 Soft tissue mass in left pontocerebellar cistern probably caused by a wide vein (→) seen at angiography (b).

as in extra axial tumours. In the second case the examination was performed in another hospital and somewhat incomplete regarding the cisterns of the posterior fossa. In both cases vertebral angiography was performed. Definite arterial changes allowing the diagnosis of an extra axial tumour or tumour vessels cannot be seen even in retrospect. There is however a displacement of the veins of the kind seen in extra axial tumours (Fig 3 b). The petrosal vein and its tributaries are elevated.

In one case of a very large acoustic neurinoma the tumour was considered to be probably intra axial. The tonsils were herniated and all the cisterns of the

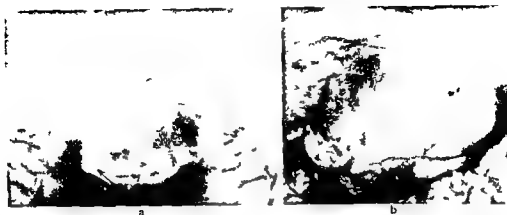


Fig 3 a) Asymmetric pontine glioma. Medullary cistern widened on tumour side with tumour pole (→) in its lateral part simulating extra axial lesion b) Local distortion of 4th ventricle indicating intra axial position of tumour

posterior to a were apparently compressed and could not be gas-filled. The ventricular system did not fill and ventriculography was not performed. A review of the vertebral angiography revealed a displacement of the petrosal vein in the same way as in the two meningiomas described but definite arterial changes can still not be seen (Fig 4). In one case of a clinically suggested acoustic tumour a soft tissue mass was seen in the pontocerebellar cistern at encephalography. Surgical exploration was however negative. When comparing the encephalograms with films from the vertebral angiography it seems evident that the soft tissue mass was probably caused by a wide vein (Fig 5).

In the remaining 3 cases asymmetrically located pontine tumours were growing into and partly filling the pontine angle cistern. In one of them the pontocerebellar cisterns were not gas-filled. The medullary cistern on one side was widened and there was an indentation from above (Fig 6). In the other two cases the pontocerebellar cistern was not demonstrated on the tumour side and was compressed on the contralateral side. Thus the changes of the cisterns were compatible with an extra axial tumour. In the first mentioned case and one of the others however ventriculography was performed and demonstrated irregular indentations in the fourth ventricle. In the third case in which vertebral angiography as well as ventriculography was performed displacement of veins of the kind seen in pontine tumours can be seen in retrospect. The fourth ventricle was displaced and flattened (Figs 7-8).

In 3 cases we consider it not possible, not even in retrospect, to make a correct diagnosis. In one of them found at surgery to have an asymmetric pontine tumour growing in the pontocerebellar cistern encephalography was performed



Fig 7 Asymmetric pontine glioma. Left vertebral angiography (subtraction film of the venous phase). Left posterior mesencephalic vein (→) displaced outwards. Petrosal vein (↔) in normal position.

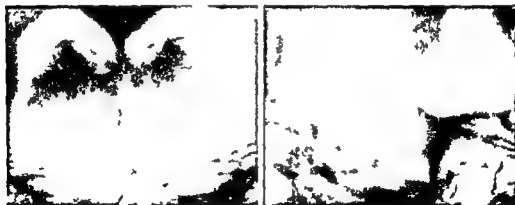


Fig 8 Same case as in fig 7. Ventriculography. Fourth ventricle displaced and flattened.

in another hospital. The cisterns of the posterior fossa were well filled with exception of the pontocerebellar cistern of the tumour side. Only some narrow streaks of gas were seen above the apex of the petrous bone, interpreted as lying in a deformed and displaced cistern. The fourth ventricle appeared normal apart from slight flattening of the lateral recess. Vertebral angiography was performed from the opposite side and the veins in the tumour area were poorly filled.

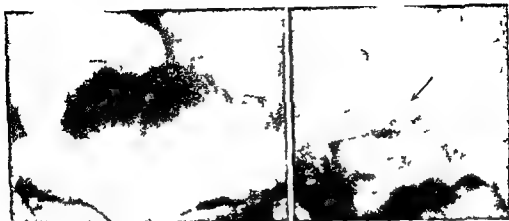


Fig 9 Ventriculography Large acoustic neuroma burrowing into the left cerebellar hemisphere Aqueduct (→) displaced anteriorly and to the right

In a second case all the cisterns of the posterior fossa were markedly compressed and no gas entered the ventricular system at encephalography. At ventriculography the aqueduct was seen to be displaced in the same way as in tumours of the cerebellar hemisphere (Fig 9). Angiography was not performed. At operation the tumour turned out to be a large acoustic neurinoma growing upward, burrowing into the cerebellar hemisphere and extending upwards through the tentorial notch which explains the changes seen. In the third case the patient had clinical symptoms and signs compatible with an acoustic tumour. At encephalography a small round soft tissue mass in the pontocerebellar cistern was interpreted as an acoustic neurinoma. At vertebral angiography only slight asymmetry of the petrosal veins was seen (Fig 10). No tumour was found at exploration. We are unable to give any explanation of the changes seen.

### Discussion

It is evident that encephalography, if performed with correct technique in most cases is a reliable method for diagnosing tumours in the pontine angle and for distinguishing between extra- and intra-axial tumours. The clinical symptoms and signs are in most extra-axial tumours fairly characteristic but may be similar in intra-axial tumours and in rare cases be lacking even in spite of large tumours. Typical bone changes can also be demonstrated in most cases of acoustic neurinomas. There are however cases in which the differential diagnosis is difficult and in some cases hardly possible to make at encephalography even if correctly performed. This is true especially in intra-axial tumours growing out into the

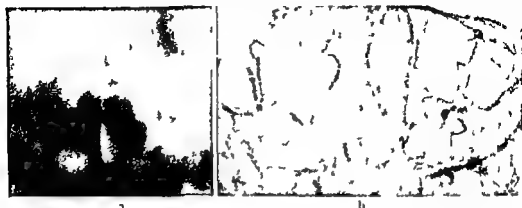


Fig. 10 a) Small soft tissue mass in left pontocerebellar cistern. b) Left vertebral angiography (subtraction film of venous phase). Slight asymmetry of petrosal veins. No tumour found at operation.

cisterns and thus partly extra cerebral, and also in extra axial tumours bulging into the brain stem and seeming to burrow into it. In these cases vertebral angiography may be helpful in the differentiation. The most characteristic changes in extra axial tumours is displacement of the petrosal vein and its tributaries best seen in the half axial a p projection. The injection should however preferably be made in the artery of the tumour side in order to secure optimal filling of the posterior inferior cerebellar artery and the corresponding veins. If for some reason the injection has to be made on the contralateral side attempts should be made to obtain a filling in retrograde direction of the vertebral artery on the tumour side by means of digital compression at its origin from the subclavian artery (Rådberg 1969) (Fig. 11).

Even though the differential diagnosis is mainly based on the appearance of the cisterns it may be necessary to take into account changes of the fourth ventricle as well. In intra axial tumours local distortions occur, which should not be mistaken for the changes caused by rotation of the fourth ventricle (cf. Fig. 2). Very small acoustic nerve tumours may also be demonstrated at cisternography, except when they are completely situated within the meatus. For the demonstration of such tumours cisternography with iodinated oil has been recommended. We have no personal experience of this method, which in our opinion could be justified only in cases in which the tumour is entirely situated within the meatus without causing evident widening of it. As far as we know we have not met with such a case. It is conceivable but unlikely that we have had a case of that kind and missed the diagnosis. If so the patient ought to have come back since all patients are followed up for a long time. In exceptional

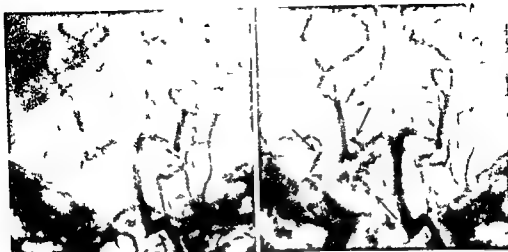


Fig 11 Left vertebral angiography Plexus papilloma of 4th ventricle After compression of right vertebral artery the highly vascular tumour (→) is demonstrated

cases it may be difficult and even impossible to obtain complete filling of the pontocerebellar cistern even when no tumour is present. In these cases vertebral angiography could help to exclude a tumour.

### SUMMARY

In order to find out with what accuracy it is possible with neuroradiologic methods to differentiate between intra and extra axial tumours in the pontine angle region a material of 150 cases has been reviewed. The differentiation is in most cases possible by means of encephalography but may be difficult and sometimes hardly possible. Vertebral angiography may be helpful in establishing the correct diagnosis.

### ZUSAMMENFASSUNG

Um festzustellen mit welcher Genauigkeit es mit neuroradiologischen Methoden möglich ist zwischen intra und extra axialen Tumoren im Winkelbereich der Brücke zu unterscheiden wurde ein Material von 150 Fällen durchgegangen. Die Unterscheidung ist in den meisten Fällen mit Hilfe der Encephalographie möglich, kann aber schwierig und gelegentlich kaum möglich sein. Die vertebrale Angiographie kann zur richtigen Diagnose nützlich sein.

### RÉSUMÉ

Pour déterminer avec quelle précision les méthodes neuroradiologiques permettent de différencier les tumeurs intra et extra axiales de la région de l'angle ponto-cérébelleux les



auteurs ont revu une série de 150 cas. La différenciation est possible dans la plupart des cas grâce à l'encephalographie mais peut être difficile et certaines fois pratiquement impossible. L'angiographie vertébrale peut être utile pour établir le diagnostic correct.

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## FORKING OF THE AQUEDUCT IN AN ADULT

by

R. OBERSON and E. GESSAGA

Pantopaque ventriculography was recently claimed to be particularly well suited for assessment of aqueductal stenosis (LANG & RUSSEL 1970). By this method however only the rostral part of the aqueduct can be demonstrated. Without demonstration of the cisterna venae magnae Galeni the length of the aqueduct is difficult to estimate. Pneumography by the lumbar route shows the caudal part of the aqueduct and the cisterns. To complete the examination a gas ventriculography can easily be performed in the same session. We can therefore not accept LANG & RUSSEL's statement that the precise point of obstruction is often not demonstrable on gas ventriculograms.

By means of tomography and the facilities offered by the rotating chair, we never failed to diagnose an aqueductal stenosis. Its type can often be assumed from a pneumogram. A rare case of posttraumatic occlusion of an aqueduct forking in an adult prompted us to review the various etiologies to be considered in such cases.

The following is a brief enumeration of the different etiologies and forms of aqueductal narrowing. For further detail the reader is referred to the pertinent literature (COLMIANT 1955, DANDY 1920, GLOBUS et coll 1942, 1946, HAMMER & PILLARI 1954, PARKER & KERNOHAN 1933, PENNYBACKER 1940, RUSSELL 1949).

*Atresia of the aqueduct*: This is a complete closure or absence of the aqueduct

This congenital malformation is incompatible with life (BICKERS & ADAMS 1949). The term atresia is also used for microscopically incomplete closure of the aqueduct. Thus, this developmental defect may vary in its degree from complete atresia to an asymptomatic minor degree, a condition incidentally revealed at postmortem examination (COLMANT 1955).

*Forking (atresia) of the aqueduct.* RUSSELL (1949) considered the term atresia incorrect since in serial sections the lumen had at no point entirely disappeared. The author defined aqueductal forking as follows: the aqueduct is represented for part of its course by two distinct channels situated in the midsagittal plane and separated from one another by normal nervous tissue. The ventral channel is usually a simple slit in the dorsoventral plane. The dorsal channel is considerably branched and the neighbouring tissue contains many groups of displaced ependymal cells, many of which form tubules. Either the dorsal or the ventral channel may unite the ventricles, the other channel dwindling and disappearing either in a cephalic or caudal direction. In some cases the two channels become fused at some point in the course of the aqueduct, and thereafter the lumen acquires a more normal aspect.

*Septum formation.* In this rare condition the caudal end of the aqueduct may be completely or partially occluded by a thin septum composed of loose textured fibrillary neuroglia (RUSSELL 1949, GRAF 1964). TURNBULL & DRAKE (1966) reviewed the subject and found 10 cases of such membranous occlusion of the aqueduct. They added and described 4 cases of their own. It is considered to be a congenital malformation or, more rarely, the result of inflammation.

*Simple stenosis* is defined as a state in which the aqueduct is histologically normal but abnormally small as compared with controls (RUSSELL). This rare condition may be inherited (BICKERS & ADAMS 1949, SAJJID & COPPLE 1968). Simple stenosis must not be confused with normal variations of the aqueduct. As WOLLAM & MILLEN (1953) have shown, the normal aqueduct varies considerably in cross-section at different levels. In fifteen normal aqueducts they found two constrictions: one at the level of the middle of the superior colliculus and the other at the level of the intercollicular sulcus. Between these constrictions the aqueduct is dilated or has the shape of an ampulla.

*Glossis of the aqueduct.* In this condition the narrowing of the aqueduct or its subdivision into two or more smaller channels is found in conjunction with a marked proliferation of subependymal fibrillary glia in the absence of inflammation, phagocytic cells or tumor elements. The narrowed lumen itself has no ependymal lining (RUSSELL). The question whether it is an acquired or a developmental abnormality is not settled. Moreover, the coexistence of fibrillary

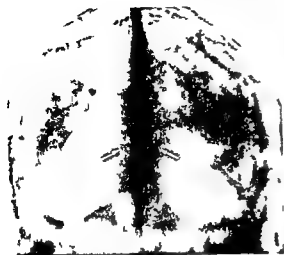


Fig 1 Carotid angiography 2 days after injury. No displacement of the thalamostriate veins ( $\rightarrow$ ) and thus no ventricular dilatation.

gliosis with other pathologic processes leads to difficulty in the separation of the various pathologic states especially when clinical history is lacking (COLMANT 1955 DRACHMAN & RICHARDSON 1961). Loss of ependymal cells as a criterion in distinguishing between developmental and acquired disorders is not always helpful, because it may be a concomitant effect of the mechanical stretching of the ependyma in hydrocephalus without inflammation. Finally, loss of ependymal cells may be a postmortem artefact (DRACHMAN & RICHARDSON).

*Inflammatory occlusion.* Acute bacterial and non bacterial meningitis may be accompanied by generalized ependymitis filling the aqueduct with inflammatory cells and debris. In chronic stages the only remnants of earlier inflammation may be in the form of disseminated ependymal granulations. But this granular ependymitis is by no means specific and may also be the result of hemorrhage surgery or other pathologic process (RUSSELL). Rarely granular ependymitis may be so considerable as to narrow the Sylvian aqueduct and lead to obstructive hydrocephalus (ADAMS & SIDMAN 1968 RUSSELL).

*Primary and secondary tumors* may obstruct the aqueduct directly or by means of compression and distortion. Tumors are usually acquired lesions but there are conditions where tumors arise on a congenital ground (COLMANT 1955 GLOBUS et coll 1942 1946 ZULCH 1948). Naturally tuberculoma, gummata and parasitic cysts may behave like tumors. A small aneurysm may also occlude the aqueduct (DAVID et coll 1951 ROWBOTHAM 1938). An angiomatous malformation of the Sylvian aqueduct with occlusion has been reported (GRAF 1943).



Fig 2



Fig 3

Fig 2 Encephalography via the lumbar route 29 days after the injury. Stenosis of the aqueduct.

Fig 3 Gas ventriculography a few days later. Marked hydrocephalus. Dilatation of the lateral part of the occluded aqueduct. The arrow points to the occlusion.

*Occlusion from pressure* by an intracranial tumor is another possibility (PARKER & KEROVIAN 1933, PENNYBACKER 1940) as well as pressure from a tentorial herniation.

### Case report

A 56-year old man was well until he was injured in an accident. Roentgen examination revealed traumatic fractures of the left parietal bone extending into the petrous pyramid. There was also a fracture of the left radial bone. During the subsequent course the patient showed persistent disturbance of consciousness. He developed marked dilation of the third and lateral ventricles and became progressively demented. Fifteen days before death a ventricular shunting was performed. However the condition of the patient deteriorated and he expired 62 days after the accident from pneumonia and urinary tract infection.

*Neuroradiologic investigation.* At carotid angiography performed 2 days after admission the ventricular system was found to be of normal size. There was no displacement of the thalamostriate veins (Fig 1).

Pneumoencephalography via the lumbar route 29 days after the injury demonstrated a stenosis of the aqueduct (Fig 2). A few days later gas ventriculography through a permanent ventricular catheter with Rickham's reservoir confirmed the aqueductal stenosis (Fig 3). The end of the aqueduct seemed to be occluded by a membrane but on the tomogram a slight basal bulging was seen (Fig 3). The two preceding tomograms (Figs 2 and 3) were superimposed in Fig 4. By this photographic method it was then possible to recognize that the exact localization of the occlusion of the aqueduct was at the level of the inferior



FIG. 4 Addition of the two tomograms in figs 2 and 3 allowing an exact localization of the occlusion ( $\rightarrow$ ) of the aqueduct. The upper part of the aqueduct is in white and the lower part and the 4th ventricle are in black.

colliculi and that there was a little shift between the rostral dorsal part and the caudal ventral part. Thus from a neuroradiologic standpoint we could relate it to a forking of the aqueduct.

**Pathologic findings** The brain appeared normal in size and shape. The temporal lobes presented old brownish discolored contused areas. The meninges and dura in these areas were thickened and brownish discolored. Beyond these areas the leptomeninges were thin and transparent. The foramina of Magendie and Luschka were patent. Frontal sections of the cerebral hemispheres revealed marked symmetric dilatation of the third and lateral ventricles. The choroid plexuses and the ependyma were smooth everywhere. The aqueduct of Sylvius was dilated rostrally but almost completely obliterated at the level of the middle of the inferior colliculi. The transition to the fourth ventricle had normal shape.

**Microscopic examination** Six  $\mu$  serial sections of the midbrain stained with hematoxylin and eosin were examined. The narrowing of the Sylvian aqueduct started at the level of the trochlear nuclei and ended almost 1 mm before the fourth ventricle. This part of the aqueduct was represented by two channels situated in the midsagittal plane. The ventral channel ended in a rostral direction at the level of the trochlear nuclei. Here the two channels were separated by normal nervous tissue (Fig 5a). At successive caudal sections both channels lined with an intact ependymal epithelium were separated and surrounded by abundant loose textured fibrillary glial tissue containing some groups of displaced ependymal cells. There were also two small ependymal tubules one of which communicated with the dorsal main channel (Fig 5b). No inflammatory phagocytic or tumor elements could be found within this fibrillary glial tissue. Still more caudally the two channels became fused (Fig 5c) and thereafter the aqueduct acquired a more normal aspect. Rostrally to the narrowing the aqueduct was dilated and rounded and showed interruption of the ependymal lining at several places. There was no granular ependymitis in the third and lateral ventricles nor in the fourth ventricle where the ependymal lining was normal. Coronal sections of the right temporal region showed a well demarcated partly cavitated cortico-subcortical contusion area surrounded by reactive astrocytes. There were numerous foamy and hemosiderin-laden macrophages around the remaining necrotic parenchyma. This lesion was quite compatible with the 17-month history following the head injury.

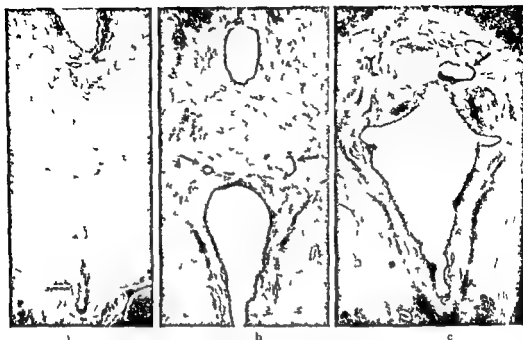


FIG. 5 Low power photomicrograms of serial sections through the midbrain (Hematoxylin and eosin  $\times 32$ ). a) Forking (arrows) of the aqueduct at the level of the trochlear nucleus. The channels are separated by normal nervous tissue. The caudal channel ends at this level ( $\rightarrow$ ). Mild ependymal gliosis around the rostral channel. b) Section 600  $\mu$  caudal to (a). Both channels are lined by flattened ependyma. They are surrounded and separated by abundant loose textured glial tissue. At the periphery there are some ependymal cell nests. Two small accessory tubules are seen within the glial bridge ( $\rightarrow$ ). c) Section 300  $\mu$  caudal to (b). The extremely narrowed rostral channel ( $\rightarrow$ ) just before the fusion with the caudal channel.

### Discussion

In our case the aqueductal narrowing was undoubtedly the cause of the dilatation of the third and lateral ventricles. The fourth ventricle was normal and its foramina were patent. The subarachnoid spaces at the base of the brain were normal. At the level of the middle of the inferior colliculi the narrowing fulfilled the criteria of aqueductal forking defined by RUSSELL. The aqueduct was subdivided into two channels and separated by normal nervous tissue (Fig. 5a), but caudally the two channels always lined by an intact ependyma were separated and surrounded by fibrillary gliosis (Fig. 5b, c). These different conditions forking and gliosis therefore coexist and a clear distinction is not easy to make. The view that aqueductal forking is a congenital abnormality is universally accepted (COORMANT) whereas the pathogenesis of aqueductal gliosis is still obscure (DRACHMAN & RICHARDSON). The patient's age (56 years),

his normal head circumference and the fact that the lateral ventricles were not dilated in the angiographic examination 2 days after the head injury indicate that the aqueductal malformation was at least until then an symptomatic one. Hence, the dilatation of the third and lateral ventricles had developed after the injury. Almost a complete stenosis of the Sylvian aqueduct was detected 29 days after the head injury by lumbar encephalography (Fig. 2). The exact situation of the lateral ventricles could be shown in the second month after the injury by cerebral pneumography (Fig. 3). RUSSELL has pointed out that whereas the ependyma is highly vulnerable and has little regenerative power, the subependymal glia is readily stimulated to proliferation. Histologically it is impossible to decide whether the fibrillary gliosis is developmental or acquired (DRACHMAN & RICHARDSON). In this case the clinical history speaks in favor of the latter. Since the fibrillary gliosis was found only at the level of the aqueductal narrowing it may be argued that the preexisting aqueductal malformation was the locus minoris resistentiae where gliosis had developed following the head injury. The report of this case is of interest because there is at least suggestive evidence that a cerebrocranial trauma was leading to functional decompensation of a preexisting congenital narrowing (forking) of the Sylvian aqueduct by fibrillary gliosis. Neuroradiologically the development of the ventricular dilatation during the clinical course and the exact situation of the aqueductal stenosis could be shown and correlated with the pathologic findings. The age of our patient could be surprising but the case described by DRACHMAN & RICHARDSON concerned a 72 year old woman. We know from RUSSELL who reported 10 cases without telling their age that forking of the aqueduct is compatible with long survival and minimal hydrocephalus should one of the channels be sufficiently well developed.

## SUMMARY

A case of posttraumatic occlusion of an aqueduct forking in an adult is documented. Lumbar encephalography and gas ventriculography are shown to be the best methods for a detailed demonstration of an aqueductal stenosis. The differential diagnosis of aqueductal occlusions is reviewed.

## ZUSAMMENFASSUNG

Ein Fall von posttraumatischem Verschluss einer Aquaduktgabelung bei einem Erwachsenen wird belegt. Es wird gezeigt, dass die lumbale Encephalographie und Gas Ventrikulographie die besten Methoden für eine detaillierte Darstellung einer Stenose des Aquadukts sind. Es wird eine Übersicht über die Differentialdiagnose für den Verschluss des Aquadukts gegeben.



## RÉSUMÉ

Les auteurs présentent un cas d'obstruction post-traumatique d'un aqueduc dédouble chez un adulte. L'encephalographie lombaire et la ventriculographie gazeuse se révèlent être les meilleures méthodes pour la mise en évidence détaillée d'une sténose de l'aqueduc. Les auteurs passent en revue le diagnostic différentiel de la sténose de l'aqueduc.

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## ANATOMISCHE DETAILS IN DER MITTELLINIE BEIM BALKENMANGEL

VON

F. P. PROBST

Es gibt zwei grosse Gruppen von komplexen Missbildungen des Gehirns die auf eine Störung der Anlage zurückgeführt werden können nämlich die sog. Callosumagenesien (REIL 1812) und die Holoprosencephalien (DE MYER & ZEMAN 1963 Synonyme Arhinencephalie KUNDRAT (1882) Holotelencephalie YAKOVLEV (1959)).

Die encephalographischen Kriterien der Callosumagenesien (DAVIDOFF & DYKE 1934 HYNDMAN & PENFIELD 1937) seien nachfolgend in der Zusammenstellung von DAVIDOFF & DYKE aufgezählt (Tabelle). Mit Ausnahme des Zeichens der radierenden Anordnung der medialen Sulci — welches allerdings ohne Tomographie im Sitzen schwer und bei fehlender Gasfüllung des Hemisphärenspaltes überhaupt nicht auszuwerten ist — ist keines dieser Kriterien für sich allein genommen beweisend ihr Wert liegt vielmehr in ihrer Kombination. Für die exakte Diagnostik gewisser Grenzfälle und solcher mit komplizierterem Erscheinungsbild ist eine sehr detaillierte Analyse der zugrundeliegenden Strukturen basierend auf gründlichen Kenntnissen der Embryologie und pathologischen Anatomie dieser Missbildungen erforderlich.

Die für die encephalographische Diagnose wesentlichen Veränderungen liegen in bzw. an der Mittellinie. Es sind dies erstens die Struktur der medialen Wände der Seitenventrikel und zweitens Veränderungen die den 3. Ventrikel betreffen.

## Tabelle

*Encephalographische Kriterien der Callosum agenesien*

## Kommentar

## Seitenventrikel

- |                                  |  |
|----------------------------------|--|
| 1) Separation                    | Kann gelegentlich durch ein nichtkommunizierendes Crura septi pellucidi et Vergae bedingt sein |
| 2) Spitze dorsale Winkel         | Nicht immer vorhanden. Können gerundet sein, besonders bei assoziiertem Hydrocephalus          |
| 3) Konka mediale Begrenzung      | Diese kann manchmal gerade oder sogar konvex sein  |
| 4) Erweiterung der kaudalen Teil | Können völlig normal sein  |

## Foramina Monro

- |               |   |
|---------------|---|
| 1) Elongation | Die Länge der Interventrikularforamina ist abhängig vom Grade der Separation. Die medialen Wände der Seitenventrikel konvergieren gelegentlich bis dicht an den erweiterten 3. Ventrikel, wobei eine Verlängerung in axialer Richtung zu mindest vorne fehlen kann. Eine wahrscheinlich in gleicher Häufigkeit vorkommende Veränderung ist die Erweiterung, also die Vergrößerung des Querschnittes dieser Kommunikation. |
|---------------|---|

## Dritter Ventrikel

- |                    |  |
|--------------------|--|
| a) Dorsalextension | a) Die Erhöhung des Daches des 3. Ventrikels kann gelegentlich uncharakteristisch sein oder fehlen.          |
| b) Erweiterung     | b) Die Erweiterung der interthalamischen Portion des 3. Ventrikels kann uncharakteristisch sein oder fehlen. |

Gehirnoberfläche  
mediale Wände

- |  |  |
|--|--|
| 7) Radiale Anordnung der medialen cerebralen Furchen und Windungen | Soweit bekannt immer vorhanden. Oft schwer nachweisbar, besonders bei der Ventrikulographie auf Grund der dabei meistens fehlenden Gasfüllung der entsprechenden Subarchnoidalräume. |
|--|--|

Das klassische anatomische Bild des kompletten Balkenmangels wird in der Abb. 1 illustriert. Das Telencephalon ist regelrecht geteilt. Die Teilung betrifft in vollkommener Weise auch die mediobasalen Teile der ursprünglichen Hemisphärenblaschen aus welchen die basalen Ganglien hervorgegangen sind und auch das Diencephalon. Dies ist bemerkenswert im Hinblick auf die später zu

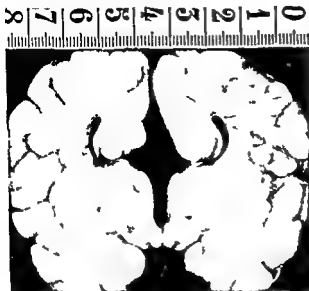


Abb 1 Acallosales Gehirn Frontalschnitt durch Corpora mamillaria Abnorme Gyrierung Gyrus cinguli beiderseits nach lateral eingerollt Sulcus cinguli fehlen Seitenventrikel symmetrisch lateral verschoben und schlitzförmig verengt Balkenlangsbündel schlank lateral buchtend Membranen den Blättern des Septum pellucidum entsprechend verbunden jederseits den Fornix mit dem Langsbündel Liquorraum zwischen Gyrus cinguli und Langsbündel = Langsbündelzisterne

besprechende Abgrenzung der teratologischen Einheit der Callosumagenesien von jener der Holoprocencephalien. Die Seitenventrikel sind nach lateral verdrängt und von medial her in typischer Weise eingebuchtet. Den medialen Abschluss der Seitenventrikel bilden jene am Querschnitt kommaförmigen langsverlaufenden Bänder welche seit 1901 (M. PROBST) als Balkenlangsbündel bezeichnet werden (longitudinal callosal bundle Probst's bundle). Die Fornices sind in der Regel vorhanden und direkt am freien Rand der Langsbündel befestigt. Gelegentlich können wie im Präparat der Abb. 1, ein oder beidseitig zwischen den Fornices und den zugespitzten Rändern der Langsbündel dünne Membranen eingeschaltet sein welche durchaus den lateralverschobenen Blättern des Septum pellucidum entsprechen. Das Dach des 3. Ventrikels ist an diesem Präparat nicht zu sehen es wurde eingerissen. Die Balkenlangsbündel und ihre Relation zu den Fornices sind von so grundlegender Bedeutung im Hinblick auf die Diagnose und systematische Einordnung dieser Missbildungen in ein teratologisches System dass auch in diesem Zusammenhang eine historische Reflexion erlaubt sein mag.

Es ist nunmehr allgemein anerkannt dass die Langsbündel überwiegend von heterotopen Balkenfasern gebildet werden welche anstatt den Interhemisphärenspalt der Quere nach zu überbrücken umgebogen und in sagittaler Richtung gewachsen sind. Diese Ansicht kann nun als bewiesen angesehen werden umso mehr als es gelungen ist den direkten Übergang der Balkenfasern vom hinteren Ende des Balkenrudimentes in die Langsbündel nachzuweisen.



Abb. 2 Ageneia corporis callosi  
Encephalogramm Langsbündel durch  
Cas in der entsprechenden Zisterne  
deinmarkiert

(M. PROBST DE LANGE 1925 LOFSER & ALVORD 1968) Das Einfließen der Langsbündelaxone in das Tapetum war schon von den älteren Autoren beobachtet und beschrieben worden. Der Verfasser dieser Arbeit ist selbst im Besitz eines solchen Präparates sowie von Encephalographiebildern, in welchen der direkte Konnex von Balkenrest und Langsbündel einwandfrei nachgewiesen werden kann.

Ein Blick auf Abb. 1 ruft ungezwungen den Eindruck hervor, dass es sich bei den Langsbündeln um die nach unten abgesunkenen und lateralverschobenen langgeteilten Hälften des Balkens handelt. Diese Ansicht wurde schon 1875 von KNOX aus Edinburgh intuitiv vertreten und anschaulich beschrieben.

This was taken to represent one half of the septum pellucidum carried away from the middle line by the divided fornix and corpus callosum. Dass diese einfache Erklärung später verworfen oder überhaupt nicht in Betracht gezogen wurde, ist auf den Umstand zurückzuführen, dass diese Strukturen keineswegs querverlaufende Nervenfasern enthielten wie zu erwarten gewesen wäre, sondern längsverlaufende. Als Konsequenz dieser Beobachtung wurde die Diskussion hauptsächlich um die Frage geführt, welches der normalen fronto-occipitalen Assoziationsbündel dieser Region eigentlich das Langsbündel bildet. Das strittige Tapetumproblem dürfte weentlich zu der entstandenen Konfusion beigetragen haben.



Abb 3 Agnesia corporis callosi. Frontale Tomographieschnitte durch das Foramen Monroi (a) und dahinter (b) Cella media von medial eingebuchtet. Längsbündelsterne nicht m. gefüllt. Rechter Fornix (—>) in lateraler Position

Der Vergleich des anatomischen Querschnittsbildes der Abb 1 mit dem Encephalogramm eines ähnlichen Falles (Abb 2) zeigt dass bei ausreichender Gasfüllung der äußeren Liquorraume die Längsbündel direkt nachgewiesen werden können. In diesem Fall sind sie annähernd keilförmig leicht gebogen mit konvexer lateraler Fläche. Darauf ist zum Teil das Zeichen der medial konkaven Wände der Seitenventrikel zurückzuführen. Diese Form der Seitenventrikel ist oft zu finden ist aber keineswegs obligat. Wenn es vorhanden ist kann es manchmal sehr ausgeprägt sein was besonders an frontalen Tomographieschnitten ersichtlich ist. In den in Abb 3 gezeigten Tomographieschnitten eines anderen Patienten sind die Seitenventrikel zu schmalen schelförmigen Spalten umgewandelt. Es ist wahrscheinlich dass diese Form in erster Linie auf sehr dicke Längsbündel bezogen werden kann. Die Dicke der Längsbündel wechselt von Fall zu Fall sie sind vorne meist dicker als hinten, können aber gelegentlich außerordentlich dünn sein.

Die zweite der für die Diagnose wichtigen Mittellinienstrukturen ist der 3. Ventrikel. Es ist ein Vorzeichen geworden dass das Dach des 3. Ventrikels beim kompletten Balkenmangel immer und beim partiellen zumindest hinten erhöht sein soll. Es wird angenommen dass das Corpus callosum, wenn es sich nach hinten über das Zwischenhirn hinwegschleibt dieses dabei nach unten drückt, mit anderen Worten dass der Descensus des Zwischenhirndaches von der Entwicklung des Corpus callosum abhängig ist. Es kann hier nicht näher auf den

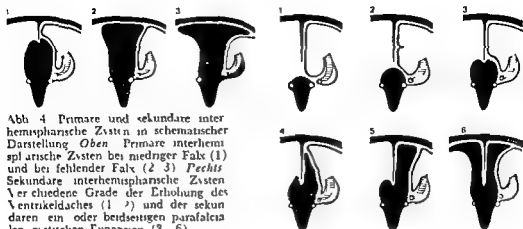


Abb. 4 Primäre und sekundäre interhemisphärische Zysten in schematischer Darstellung. Oben: Primäre interhemisphärische Zysten bei niedriger Falx (1) und bei fehlender Falx (2, 3). Pechts: Sekundäre interhemisphärische Zysten. Verschiedene Grade der Erhöhung des Ventrikeldaches (1, 2) und der sekundären ein- oder beidseitigen parafalcialen zystischen Expansion (3—6).

Mechanismus der Verschiebung der verschiedenen Hirnstrukturen im Laufe der Entwicklung eingegangen werden, doch scheint klar zu sein, dass die Senkung eine Folge des unterschiedlichen Wachstums der verschiedenen Hirnteile ist. Auch die transverselle Rotation der Thalami bewirkt eine Senkung des 3. Ventrikels und seines Daches, bevor das Callosum überhaupt existiert. Im übrigen ist auch die Bildung des Velum interpositum eng mit den Relationen zwischen Balken und Zwischenhirndach verknüpft.

Eine Erhöhung des Daches des 3. Ventrikels ist wirklich in der Mehrzahl der Fälle vorhanden, doch gibt es Ausnahmen. Diese Erhöhung ist von sehr verschiedener Grösse und schwankt von einer kaum merkbaren Elevation bis zur Bildung gross aufgeblauhter Zysten.

Gerade die Betrachtung der dorsalen Zysten gibt uns Hinweise auf den Mechanismus der Senkung des Zwischenhirndaches im Laufe der Entwicklung. Wenn man die Verhältnisse etwas näher analysiert, ergibt sich, dass es zwei prinzipiell verschiedene Situationen gibt. Wie aus den Skizzen der Abb. 4 zu sehen ist, existieren einerseits Zysten in deren Bereich die Falx cerebri fehlt und andererseits solche bei normal ausgebildeter Falx.

Bei den Formen der ersten Gruppe liegen die Zysten in der Mittellinie und drängen die Hemisphären nach lateral ab. Sie können sich nach oben bis an die Konvexität erstrecken, gegebenenfalls die Mantelkante umgreifen und sich nach hinten bis zum Tentorium ausdehnen. Betrachten wir die zweite Gruppe, so finden wir, dass bei geringer Elevation das Ventrikeldach die Falx nirgends erreicht. Bei grosserer Expansion dehnt sich das Ventrikeldach, das nun endgültig den Charakter einer Zyste angenommen hat, an der Falx entlang nach oben aus und zwar an einer oder beiden Seiten, mehr oder weniger



Abb 5 Encephalogramme primärer interhemisphärischer Zysten

symmetrisch. Auch diese Zysten können schliesslich grosse Teile des Interhemisphärenspaltes okkupieren.

Die regelrechte Ausbildung der Falx cerebri ist nur dann möglich, wenn die Hemisphären sich ordnungsgemäss geteilt und aneinandergelegt haben. Wird dieser Prozess durch die Existenz einer dazwischenliegenden Expansivität gestört, kann die Falx in diesem Bereich nicht gebildet werden. Die Falxdefekte der ersten Gruppe weisen auf eine solche Störung hin. Es muss angenommen werden, dass wohl die Thalami nicht, aber das Zwischenhirndach ordnungsgemäss descendiert sind. Letzteres hat also seine von Anfang an erhöhte Position zwischen den Hemisphärenblaschen beibehalten und die Bildung der Falx ganz oder teilweise verhindert, wobei die Ursache unbekannt bleibt. Diese Zysten müssen daher als primäre interhemisphärische Zysten bezeichnet werden. Sie entstehen früher als die sekundären und es ist wahrscheinlich, dass sie nicht nur die Bildung der Falx, sondern auch die des Corpus callosum verhindern können. Solche Zysten können auch bei partiellen Callosumagenesen vorkommen, wobei sich der vordere Balkenrest bis an die vordere Zystenwand erstreckt.

Die Zysten der zweiten Gruppe sind demnach als sekundäre interhemisphärische Zysten zu bezeichnen. Die Falx ist dabei intakt. Das bedeutet, dass diese in ihrer normalen Ausbildung nicht behindert worden ist und auch, dass das Zwischenhirndach vorher zumindest bis unter das Niveau der Falxklante





Abb 6 Encephalogramme sekundärer interhemisphärischer Zysten

abgesunken sein muß. Es beweist weiterhin, dass der Deszensus des Zwischenhirndaches ein normaler Vorgang ist, der mit der Expansion des Balkens sehr wenig zu tun hat, weil dieser schon zu einem Zeitpunkt erfolgt, wo der Balken noch nicht existiert. Zusammenfassend kann gesagt werden, dass die primären Zysten das Resultat eines nicht stattgefundenen Deszensus, die sekundären aber das einer sekundären Ausblähung des Zwischenhirndaches sind. Die Abbildungen 5 und 6 zeigen je zwei primäre und sekundäre interhemisphärische Zysten.

Es kann nun gewiss nicht jeder Einfluss des Balkens auf die Stellung des Zwischenhirndaches geleugnet werden. Dieser Einfluss ist jedoch indirekt und das hängt mit den Vorgängen bei der Bildung des Velum interpositum zusammen. Er dürfte auch relativ beschränkt sein, auch wäre den Fornices dabei eine bedeutendere Rolle zuzuschreiben als dem Callosum selbst, welches ja bestenfalls nur mit der oberen Wand des Recessus suprapinealis in direkten Kontakt tritt. Immerhin kann gesagt werden, dass die Abwesenheit des Callosum und in noch höherem Grade die Separation der Fornices die sekundäre Dorsalexpansion des Zwischenhirndaches ermöglicht.

Die Anatomie der Mittellinienstrukturen ist von grosser Bedeutung auch für das Problem der Abgrenzung der Formen des Balkenmangels von jenen der Holoprosencephalie. Es herrscht seit langem die Ansicht, dass die meisten oder zumindest gewisse Typen von Balkenmangel zum Formenkreis der Holoprosencephalien zu rechnen seien, nämlich die mit assoziierter Arhinencephalie und jene mit dorsalen (interhemisphärischen) Zysten. Diese Auffassung kann bis ins Jahr 1882 zurückverfolgt werden, als KUNDRAT seine bekannte Arbeit

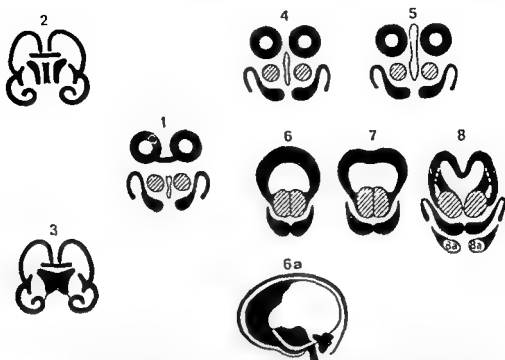


Abb 7 Schematische Klassifikation der komplexen durch Anlagestörung bedingten Missbildungen des Gehirns (1) Symbol für das normale Gehirn (2) Cavum septi und Vergae (3) Agenesie des Septum pellucidum (4) und (5) Zwei Formen des kompletten Balkenmangels (6) (7) und (8) Verschieden hoch entwickelte Formen des Spektrums der Holoprosencephalie

über die Arten der Arhunencephalie schrieb Sie wird auch heute noch vertreten (CURRARIO & SILVERMAN 1960 DE MYER & ZEMAN 1963 ZINGELSSER et coll 1966 u a )

Es liegt sicherlich kein Grund vor einen typischen Fall von Callosumagenesie auf Grund einer übrigens äußerst seltenen assoziierten Arhunencephalie (Fehlen der Bulbi und Tractus olfactorii) als Holoprosencephalie anzusprechen. Das Gesagte gilt auch für die Callosumagenesen mit interhemisphärischen Zysten. Es ist nicht gerechtfertigt die teratologische Einheit dieser Missbildungen auf Grund eines Analogiechlusses anzunehmen. Die Zysten der Callosumagenesen sind nämlich keineswegs mit jenen der primitiven Formen der Holoprosencephalie gleichzusetzen. Die dorsalen Zysten der Callosumagenesen liegen zwischen den Hemisphären, was die regelrechte Teilung des Vorderhirns voraussetzt. Die dorsalen Zysten der Holoprosencephalien dagegen sitzen der (ungeteilten) Holosphäre hinten auf. Wenn sie sich aber was gelegentlich vorfindet zwischen den halbgeteilten Hemisphären in der Mittellinie nach vorne

erstrecken sind sie nicht wie bei der Callosumagenesie an den Langsbundlestrukturen bzw. den daran fixierten Fornices befestigt, sondern an den dorsal liegen gebliebenen Hippocampi. Dieser Unterschied ist wesentlich, kann aber bei der Encephalographie ein differentialdiagnostisches Problem darstellen. Der Zeitpunkt für die Evagination der Hemisphärenblaschen bildet die Grenzlinie zwischen den Terminationsperioden der Holoprosencephalien und Callosumagenesien (Fetalalter 30—32 Tage, Horizon XV nach Streeter, Scheitel-Steißlänge 7—8 mm). Diese Grenze ist so zu verstehen, dass die Missbildungen des Holoprosencephaliespektrums nicht nach dem erwähnten Zeitpunkt terminiert werden können, wogegen eine eventuelle Termination der Callosumagenesien vor diesem Zeitpunkt nicht ausgeschlossen werden kann. Ob letzteres die Regel oder die Ausnahme darstellt ist nicht bekannt.

Es gibt noch weitere Gründe für die Behauptung, dass die Callosumagenesien auf keinen Fall mit den Holoprosencephalien in einen Topf geworfen werden dürfen. Darauf kann im Rahmen dieser Arbeit nicht eingegangen werden; diese Frage wird in einer kommenden Publikation eingehend besprochen werden. Hier soll nur noch hinzugefügt werden, dass eine systematische Klassifikation der Missbildungen dieser beiden Gruppen strikt an der pathologischen Anatomie orientiert werden muss. Entfernte encephalographische Ähnlichkeiten, die Neurologie oder die mentalen Defekte verschiedenen Grades und auch das Nichtvorhandensein des Corpus callosum *per se* sind keine ausreichenden Kriterien für eine distinkte Definition dieser Zustände (vgl. das Schema von CASTROVIEJO 1969).

Ein einfaches Schema, welches die markanten Unterschiede dieser Missbildungen illustriert, ist in Abb. 7 gegeben. Nr. 1 ist das normale Gehirn. Die Ringe symbolisieren die normalen, geteilten Hemisphären, der Querbalken das Corpus callosum. Darunter die geteilten Thalami und zwischen ihnen ein normaler 3. Ventrikel. Darunter die geteilten Striata. Links oben (Nr. 2) ein Gehirn mit einem Cavum septi und Vergae (eigentlich keine Anlagestörung, sondern nur die Persistenz eines passageren embryonalen Zustandes). Links unten (Nr. 3) ein Gehirn mit Septum pellucidum aplasie. Rechts oben (Nr. 4, 5) zwei Formen der Callosumagenese, die sich nur durch die Größe der interhemisphärischen Zyste unterscheiden. An Stelle des Callosum finden sich die beiden Langsbündel. Diese Gehirne sind ansonsten anlagemässig intakt. Dass die Anlage (das Gehirn) in sekundärer Weise in verschiedener Kombination und verschieden schwer gestört sein kann, ist eine andere Sache und spielt für diese prinzipielle Klassifikation keine Rolle. Rechts unten (Nr. 6, 6a, 7, 8) die Symbole für drei Formen der Holoprosencephalie. Nr. 6 und 6a zeigen die primitivste Form mit ungefalteter Holosphäre, verschmolzenen Thalami und Striata. Das Seitenbild (Nr. 6a) illustriert die Position des Gehirns

im vorderen Teil der Schadelkapsel und die große dorsale Zyste (the dorsal sac) Nr 7 und 8 symbolisieren graduell höher differenzierte Formen mit größerer Gehirnmasse die zu einer eichten (Nr 7) und tiefen (Nr 8) aber vorne unvollständigen Einfaltung des Gehirnmantels geführt haben. Bei der letztgenannten Form sind die Thalami geteilt der dazwischen liegende Ventrikel ist ein Teil des Monoventrikels Die Striata sind immer noch ungeteilt können aber gelegentlich auch geteilt sein

### Konklusion

Die anatomischen Verhältnisse in der Mittellinie sind von entscheidender Bedeutung für die distinkte Klassifizierung der komplexen Anlagestörungen des Gehirnes Die Balkenlangsbündel sind die für die Callosumagenesen charakteristischen Strukturen Sie kommen bei keiner anderen Form von Missbildung vor Voraussetzung für ihre Existenz ist die komplette Teilung des Endhirns in Hemisphären Der zweite Punkt von Bedeutung ist das Verhalten des Daches des 3 Ventrikels Die dorsalen Zysten der callosumagenesen sind interhemisphärische Zysten und können nach ihren Relationen zur Falx in primäre und sekundäre unterteilt werden Die primären Zysten entstehen zu einem früheren teratologischen Zeitpunkt und sind die Folge eines nicht stattgefundenen Deszensus des Zwischenhirndaches Die sekundären Zysten entstehen zu irgendeinem späteren Zeitpunkt der totalen Entwicklung und wahrscheinlich auch post natum Die Formen der Holoprosencephaliegruppe zeichnen sich durch eine bestenfalls in der Mittellinie eingefaltete Holosphäre aus Sie besitzen weder ein Corpus callosum noch Balkenlangsbündel Die dorsalen Zysten der primitiven Formen dieser Gruppe unterscheiden sich grundlegend von den interhemisphärischen Zysten der Callosumagenesen Es gibt daher keinen Grund dafür die letzteren den Formen der Holoprosencephalie zuzurechnen, genau so wenig wie die zufällige Koexistenz einer isolierten Arhinencephalie mit einer Callosumagenese die als Holoprosencephalie qualifiziert Callosumagenesen und Holoprosencephalien sind strukturell wenn auch nicht bezüglich der Terminologie streng voneinander geschieden Die Unterscheidung kann nicht nur autopsisch sondern auch durch das detaillierte Studium der Anatomie mit Hilfe der Encephalographie durchgeführt werden

### ZUSAMMENFASSUNG

Die Grundzüge der Anatomie der Mittellinienstrukturen beim Balkenmangel werden beschrieben und ihr Wert für die Klassifizierung dieser Missbildungen sowie deren Abgrenzung von den Formen der Holoprosencephalie dargelegt

## SUMMARY

The basic anatomic features of the midline structures in agensis of the corpus callosum are described and their value in the classification in this type of malformation and the differential diagnosis from forms of holoprosencephaly discussed

## RÉSUMÉ

L'auteur décrit les caractères principaux de l'anatomie des structures de la ligne médiane dans l'agenésie du corps calleux et montre leur intérêt pour la classification de ces malformations et pour les distinguer de certaines formes d'holoprosencéphalie.

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## THE NORMAL ENCEPHALOGRAM DURING THE FIRST TWO YEARS OF LIFE

by

H. ROSENGREN and C. A. CARLSSON

This investigation was concerned with the roentgen appearances of the ventricular system and the subarachnoid spaces with special reference to their width during the first two years of life by means of encephalography.

*Material* One hundred and fourteen children under two years old were examined during the period 1962—1965. 87 of them were alive at clinical control in 1969 i.e. 4 to 7 years later. 29 being of average mentality and normally developed. Thirteen of this group of 29 normal children were excluded either because of obvious signs of cerebral damage at the time of the primary examination or because they had had surgical treatment.

The remaining 16 children were classified as normal. The symptoms and signs that had been the indications for the encephalography had completely disappeared during their stay in hospital or shortly afterwards or had obviously had no influence on the intracranial structures, the children also had remained free from cerebral symptoms during the control period. The clinical diagnoses

in this group at the time of the encephalography are presented below

Diagnosis	No. of patients
Extracranial tumours	4
Dermoids (3)	
Lipoma (1)	
Aberrant forms of the head	8
Caput quadratum (2)	
Large head (2)	
Scaphocephalic (3)	
Unilateral bulging (1)	
Convulsions	2
Dermal defect over the thoracic spine	1
Gait disturbances	1

No signs of increased intracranial pressure were present and it was evident at operation that the tumours (3 dermoids and 1 lipoma) failed to interfere with intracranial structures. The dermal defect over the thoracic spine was superficial and did not affect the meninges. The infants with aberrant cranial forms presented no signs of cerebral dysfunction. Carotid angiography demonstrated normal conditions in those with caput quadratum (usually associated with rickets) and local bulging of the skull. The two children with convulsions had at home had two and three attacks respectively, described by the parents as convulsions. They presented no such manifestations or other signs of cerebral disorder however while they were in hospital or afterwards. The girl (aged 14 months) with gait disturbances had impairment of walking following a slight head injury; a lesion in the posterior fossa was considered probable. She improved under hospital treatment and three to four weeks after the accident behaved quite normally as she has done ever since.

*Method* The encephalography was always performed with the fractionated insufflation of oxygen after lumbar puncture with the infant sitting. FFD 100



Fig. 1. The Evans index (y axis) of the 16 infants plotted against their ages in months.

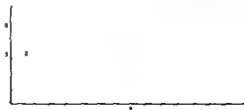


Fig. 2. The width of the third ventricle in mm (y axis) plotted against the age in months.



Fig 3 a) Ap and b) lateral projection of an infant aged one week



Fig 4 a) Ap and b) lateral projection of an infant aged one month. The arrows indicate the means of measuring the sulci



Fig 5 The widths of a representative gas filled sulcus in mm (y axis) plotted against the age in months



cm. All examinations were performed under general anaesthesia, the tube connection to the anaesthetic trolley being long enough to permit easy handling of the infants in all positions.

### Results

Oxygen always passed into the ventricular system. The width of the lateral ventricles was calculated according to the EVANS index, i.e. the ratio between the width of the frontal horns and the inner skull diameter. The normal value in adults is 0.23, ranging from 0.16 to 0.29 (EVANS 1942), the mean figure in this particular investigation was 0.30 and ranged from 0.24 to 0.33. The indices in Fig. 1 are plotted against the ages in months of the infants at the time they were examined. The width of the third ventricle varied between 2 to 8 mm with a mean of 5 mm. The relation between the width and age in months of the child appears in Fig. 2. Neither the EVANS index nor the diameter of the third ventricle seems to be correlated to the age.

The cisterna veli interposita was well filled in six examinations, otherwise the cisterns had roughly the same appearances as in adults.

The gas entered a space over the hemispheres in all cases except one in which a small amount appeared in the lateral fissure on one side only. The gas was distributed as in Figs 3 and 4 in all subjects aged between one week and 5 months; this type of gas distribution was not evident in infants older than 6 months. The depth of the space and the fact that the gas moves freely indicates its subdural collection, whereas the wavy inner contour towards the brain is in favour of the gas being subarachnoidal. The gas is probably partly inside and partly outside the arachnoid membrane. This assumption implies defects in the membrane, either preformed or more likely due to a rupture. This theory is supported by the postmortem examination of infants where the membrane appears to be thin and to lie fairly close to the brain. The width of the sulci gradually diminishes with increasing age as in Fig. 5. The measurements of the sulci have been performed in lateral films. Fig. 4b and 6b demonstrate how the measurement is performed and the width of the sulci determined. The



Fig 6 a) Ap and b) lateral projection of an infant aged 5 months. The arrows indicate the means of measuring the sulci.



Fig 7 a) Ap and b) lateral projection of an infant aged 11 months.

authors are aware of the possible bias in measuring, but the tendency is obvious and it appears that the width of the sulci does not noticeably decrease after the age of about 12 months: at this period the width is 2 to 3 mm, which can be taken as lying within normal limits in adults. The typical gas distribution over the hemispheres in children of varying ages is demonstrated in a representative series of encephalographies (Figs 3, 4, 6, 7).

### Discussion

The ventricular system as well as the extracerebral spinal fluid pathways in several respects present somewhat different appearances in infancy and early childhood than in adults. LODIN (1968) has investigated the normal topography of the cerebral ventricular system up to 15 years of age. He, however, restricted himself to measuring the position of the optic recess of the third ventricle, the aqueduct and the fourth ventricle in correlation to certain bony landmarks and did not discuss the width of the spaces. The present authors feel it is important to have some knowledge of the normal variations of this since otherwise the diagnosis of atrophy may be erroneous. The material is fairly small but strictly selected and appears to be fairly uniform and representative of the normal variations. It is interesting to recall that LODIN reported that the most rapid changes occurred during the first two years of life. The difference between adults and children was most evident during the first year of life in the present investigation.

### SUMMARY

The encephalograms of infants that had developed normally and obviously had had no disease affecting intracranial structures at the time of the investigations or later were examined in retrospect. Special attention was directed to the appearances of the gas over the hemispheres at the various ages. Their significance is discussed.

### ZUSAMMENFASSUNG

Die Encephalogramme von Kindern, die sich normal entwickelt hatten und deutlich keine Erkrankungen hatten, die die intrakraniellen Strukturen zum Zeitpunkt der Untersuchungen oder später beeinflussten, wurden retrospektiv untersucht. Besonders wurde dem Auftreten von Gas über den Hemisphären bei verschiedenen Altern Beachtung geschenkt. Dessen Bedeutung wird diskutiert.

### RÉSUMÉ

Les auteurs ont examiné rétrospectivement les encephalographies gazeuses de nourrissons qui ont eu un développement normal et n'avaient indiscutablement aucune affection des structures intracrâniennes au moment de l'examen ou plus tard. Ils ont particulièrement étudié les images gazeuses à la convexité des hémisphères aux différents âges. Ils examinent l'intérêt de ces images.

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## CISTERNAL DILATATION IN INFRATENTORIAL TUMOURS

Experimental reproduction of the probable  
pathogenetic mechanism in the cadaver

by

M ROTH J HORRICKA and I TOMAN

Dilatation of the perimesencephalic cisterns viz the quadrigeminal velum interpositum and ambient wing cisterns together with the callosal sulci may be occasionally observed during encephalography in the presence of an infratentorial tumour associated with occulsive hydrocephalus (CIBA & WENDT 1960 JACOBSON et coll 1965 LILIEQUIST 1963 PRIERAV 1963 SASSAROLI 1955 SCHECHTER et coll 1958 TAVERAS 1960 and others). The dilatation of the cisterns appears somewhat paradoxical since in obstructive hydrocephalus compression rather than dilatation of the supratentorial cisternal spaces would be expected.

Several years ago the following speculative explanation of this phenomenon was offered (ROTH 1961, 1963). Under normal conditions the cerebrospinal fluid leaves the posterior fossa through the entire circumference of the tentorial incisura (Fig 1 a). With the ascending cerebellar herniation accompanying a posterior fossa tumour the incisura becomes progressively obstructed except at the apex which constitutes its highest point, and to which the cerebrospinal fluid



Fig 1 a



Fig 2 a

Fig 2 b

Fig 1 The normal a) and presumed pathologic streaming of the CSF through the tentorial incisura in the presence of a posterior fossa tumour (b)

Fig 2 a) Three inflated balloons in the evacuated posterior fossa (the median pontile balloon is hidden by the two lateral hemispheric balloons). The incisura is obstructed except at the apex. b) The fluid injected through a catheter (left upper corner) into the posterior fossa appears at the apex.



Fig 1 b

pathway is restricted. The fluid is thus directed into the median and paramedian supratentorial cisterns which become dilated in the course of time under its steady impact (Fig 1 b).

An attempt has been undertaken to buttress the proposed hypothesis by experiments in the cadaver. Three balloons were introduced into the evacuated posterior fossa: one median or pontile and two lateral or hemispheric. When inflated the balloons blocked the incisura completely except at the apex (Fig 2 a). Methylene blue tinted water was then injected by means of a catheter inserted through the tentorium into the posterior fossa: the fluid appeared at the apex (Fig 2 b).

Fig 3 Balloon introduced at the bottom of the posterior fossa by means of a glass tube (a) represents after inflation a tumour causing upward cerebellar herniation (b).

Fig 4 The uninflated balloon in situ under normal conditions. The blue tinted fluid leaves the posterior fossa along the margins of the tentorial notch (evident on both sides of incisura). The catheter for injection of fluid runs along the infolded falx.

Fig 5 Balloon partly inflated (cf fig 3 b). The flow of the injected fluid is restricted to the apex (cf fig 4 b) and may appear even as a jet.

Fig 6 a) Massive upward cerebellar herniation completely obstructing the incisura. b) Digital compression from above frees the apex from the herniating tissue and the fluid shoots through.

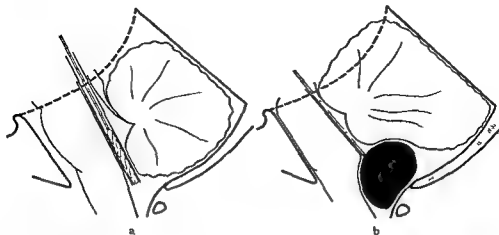


Fig 3 (For legend see opposite page)



Fig 4



Fig 6a



Fig 5



Fig 6b

(For legends see opposite page)



Fig 7

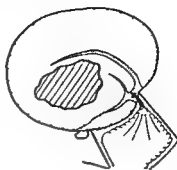


Fig 8

Fig 7 The incisura including the apex in the absence of pressure hydrocephalus is completely blocked by the unimpeded massive upward herniation of the cerebellum

Fig 8 Obstruction of the tentorial notch except for the apex from above in the presence of a supratentorial tumour may also lead to cisternal dilation

The contents of the posterior fossa were left in place and the cerebral stem was sectioned at the level of the tentorial notch in the following experiments. An infratentorial neoplasm was imulated by means of a balloon introduced via the aqueduct and the fourth ventricle at the bottom of the posterior fossa. The insertion of the balloon was facilitated by a glass tube which was then withdrawn (Fig 3). Before the inflation of the balloon i.e. as in normal conditions, the injected fluid pours out freely along the margins of the incisura (Fig 4). With as few as five to ten millilitres of air introduced into the balloon upward herniation of the cerebellum however already takes place and the incisura is more or less obstructed. The flow of the injected fluid is by reason of the slight degree of insufflation of the balloon restricted to the apex, where it may sometimes appear even in the form of a jet (Fig 5).

Only in 3 out of 15 experiments could the effect described be obtained. The injection of fluid usually led to a rapid and massive herniation resulting from the accumulation of the fluid beneath the cerebellum (Fig 6a).

Digital compression of the massively herniated cerebellum from above sometimes produced a jet of fluid from the apex which was freed from the herniating tissue by the applied pressure (Fig 6b). This phenomenon explains the indispensable effect of obstructive hydrocephalus in the production of cisternal dilatation (Fig 7). The counter pressure of the hydrocephalic brain obviates massive ascending cerebellar herniation (JEFFERSON 1931) that would result in complete obstruction of the incisura including the apex. Dynamic equilibrium is created at the level of the incisura by the pressure of the hydrocephalic cerebrum

from above and of the herniating cerebellum from below, leaving the apex free for the passage of the cerebrospinal fluid

The rather small number of experiments prevented any investigation of the possible relation of the phenomena described to the general size and shape of the tentorial notch

The cisternal appearances characteristic of infratentorial expansive processes may be observed exceptionally in supratentorial tumours as well (Fig 8) The tentorial notch may be obstructed from above again with the exception of the apex by the expanding brain tissue a situation promoting dilatation of the cisterns thus created

The experimental observations seem thus to support the explanation suggested No absolutely certain proof can of course be claimed since the experimental conditions were too remote from those existing in the living subject Further research is obviously indicated

## SUMMARY

The restriction of the cerebrospinal fluid pathway to the apex of the tentorial notch considered possible for the supratentorial cisternal dilatations in posterior fossa tumours was reproduced experimentally in the cadaver

## ZUSAMMENFASSUNG

Die Einschränkung des cerebrospinalen Flüssigkeitsweges zur Spitze des Tentoriumschlitzes die als verantwortlich für die Erweiterungen der supratentoriellen Cisternen bei Tumoren der posterioren Fossa angesehen wird wurde experimentell am Kadaver nachgebildet

## RÉSUMÉ

Les auteurs ont reproduit expérimentalement sur le cadavre le retrecissement du passage du liquide céphalo rachidien au niveau du sommet de l'incisure de la tente retrecissement qui est considéré comme la cause de la dilatation des citernes sus tentorielles dans les tumeurs de la fosse postérieure

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## NINTH SYMPOSIUM NEURORADIOLOGICUM GOTHENBURG 24-29 AUGUST 1970

### PART II

As in Part I the contributions are grouped according to the subject matter and the aspects from which this is treated. Some of the contributions have therefore not been included in the same subject groups as in the symposium programme. The classification of certain papers, such as those dealing with comparisons between different methods of examination may be a matter of opinion but we have endeavoured to place each paper under the heading that seemed most appropriate. In each group, the papers are arranged in alphabetical order according to the first author's name. The titles of the Symposium papers not published here are included in the Table of Contents under the sections in which they appeared in the Symposium programme.

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 Sjaastad O 595  
 Skalpe I O 865  
 Skoldborn H 634 643  
 Soderborg B 554  
 Steinle R 612  
 Strohecker J 842  
 Sugiura K 599  
 Sumner K 693  
 Taylor K J W 743  
 Torres J 767  
 de Tovar G 475  
 Vanselow K 905  
 Vertiy Pamela 496  
 Viale G L 590  
 Wackenheim A 875  
 Weill F 612  
 Weiss M H 615  
 Wickbom I 634 643  
 Yabe Y 599  
 Zander E 524

**HYDROCEPHALUS INCLUDING CEREBROSPINAL  
FLUID CIRCULATION**



## INDICATIONS DE LA DERIVATION VENTRICULO CISTERNALE ET CONTRÔLE DE SON EFFICACITE PAR LES RADIO ISOTOPES

par

M ALERMAN, G DE TOVAR et G GUIOT

Le traitement chirurgical des hydrocephales obstructives consiste à drainer l'excès de liquide céphalo rachidien ventriculaire. Plusieurs types d'intervention peuvent être choisis. L'intervention la plus courante est la dérivation externe avec pose d'une valve : dérivation ventriculo-atriale ou ventriculo-péritonéale. Lorsque l'obstruction siège au niveau du système ventriculaire et à condition que les espaces sous-arachnoïdiens péri-cérébraux soient perméables, une dérivation interne peut être suffisante. Parmi les méthodes de dérivation interne la ventriculo-cisternostomie, sous contrôle radioscopique, récemment décrite par GUIOT et coll est une intervention très simple et bénigne.

L'intérêt des techniques de cisternographie et de ventriculographie isotopiques proposées par DI CHIRO a été souligné par de nombreux auteurs (ALERMAN et coll, BROCKENHURST, HEINZ et coll, OBERSON et coll, SPOERRI et coll, TATOR et coll).

L'exploration isotopique renseigne sur la morphologie des cavités sous-arachnoïdiennes et ventriculaires et, surtout, donne des informations sur la dynamique circulatoire du LCR et ses échanges avec le sang.

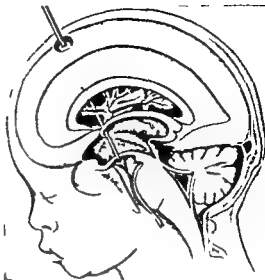


Fig. 1. Schéma de la technique de ventriculo-cisternostomie de Guiot au col. Le trocart leucotome perce le plancher du 3ème ventricule après être passé par le foramen de Monro.

Nous utilisons ces explorations topiques depuis 3 ans pour le diagnostic des hydrocephalies et pour tester le fonctionnement et l'efficacité des dérivations neuro-chirurgicales.

L'indication du mode de dérivation d'une hydrocephalie dépend, avant tout, de la perméabilité des espaces pericerebraux et des possibilités de reabsorption physiologiques du LCR. Le choix de la dérivation sera fonction des informations apportées par l'exploration topique.

### Techniques et Matériel

La *entriculo cisternostomie* est réalisée en introduisant, sous contrôle radio-télévisé, un trocart leucotome dans le 3ème ventricule en passant par voie coronale et à travers le foramen de Monro (Fig. 1). Deux ml de lipodol fluide sont injectés dans le 3ème ventricule (Fig. 2). Dans les cas favorables, le lipodol dessine une double besace de part et d'autre de la clinode postérieure; la besace antérieure bombe dans la cavité ciliaire tandis que la besace postérieure bombe dans la citerne inter pedonculaire. C'est cette poche postérieure qui est visée par le trocart et le leucotome de Claude Bertrand va perforer le plancher du 3ème ventricule, faisant communiquer ce 3ème ventricule avec la citerne inter pedonculaire. Le lipodol s'écoule alors rapidement, franchit le trou occipital et tombe dans le rachis.

Les *scintigraphies* sont réalisées à l'aide de serum albumine marquée à  $^{125}\text{I}$  de haute activité spécifique injectée dans le LCR à raison de 50 à 100  $\mu\text{Ci}$ ,



Fig 2 Controle radiologique de la perforation du plancher du 3eme ventricule dans la cistne interpedunculaire

dans un volume de 1 a 3 ml L injection est faite par voie lombaire sous-occipitale ou par ponction ventriculaire

Des scintigraphies successives ont enregistrees avec un Magnascanner III generalement 3, 6, 24 et parfois 48 heures apres l injection

*Le controle des derivations chirurgicales est realise apres injection ventriculaire du compose radio-actif En plus des images scintigraphiques on mesure la decroissance de la radio-activite ventriculaire, ainsi que l ont propose ATKINSON & FOLTZ Mais au lieu de faire des prelevements de LCR comme ces auteurs nous nous contentons de mesurer la radio-activite ventriculaire par comptage externe en disposant devant chaque ventricule lateral le detecteur collimate du Magnascanner Ces mesures ont faites une heure 3 heures et 24 heures apres injection Les activites de la 3eme et de la 24eme heure sont exprimees en pour cent de la radio activite mesuree a la 1ere heure*

De plus est mesure l accroissement de la radio-activite sanguine en disposant devant l aire cardiaque le detecteur non collimate utilise habituellement pour la mesure de la fixation thyroïdienne Les valeurs ont egalement exprimees en unites arbitraires en pour-cent de la radioactivite ventriculaire comptee a 1 heure

Cent dix explorations craniennes ont ete realisees a ce jour Vingt-quatre de ces explorations ont ete faites apres injection ventriculaire du produit radio-actif

Vingt examens ont ete faits chez 14 malades ayant subi une ventriculocisternostomie



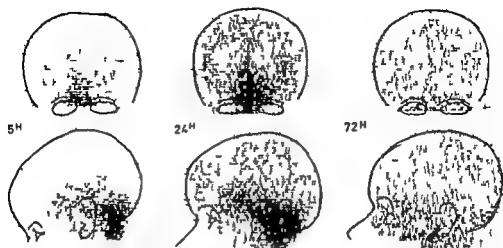


Fig. 3 Cisternogrammes préopératoires dans un cas de sténose de l'aqueduc de Sylvius. Les écoulements sous arachnoïdiens péri-cérébraux sont perméables. Il n'existe qu'un simple retard du transit radio-actif.

Fig. 4 Blocage de la radio-activité au niveau des cisternes de la base du crâne dans une hydrocéphalie par sténose de l'aqueduc associée à des troubles de la perméabilité des écoulements péri-cérébraux.



## Resultats

*L'exploration préopératoire par injection sous arachnoïdienne.* En l'absence d'hydrocéphalie nous retrouvons les images cisternographiques maintenant bien connues. Le liquide radio-actif progresse de façon régulière depuis la grande citerne jusqu'aux aires de résorption du LCR, à la convexité cérébrale. Il n'impregne pas le système ventriculaire. À la 24<sup>ème</sup> heure la plus grande partie de la radio-activité se trouve au niveau des aires de résorption de la convexité et du sinus longitudinal.

Dans les hydrocéphalies communicantes, on retrouve le signe caractéristique du reflux de la radio-activité dans les ventricules avec une absence plus ou moins complète de résorption du LCR. Dans ces cas la dérivation externe représente le seul traitement efficace.

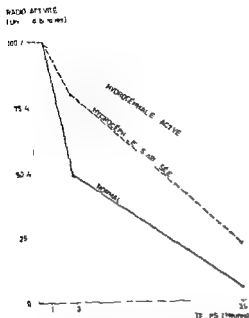


Fig 5 Courbes de décroissance de la radio-activité ventriculaire dans 3 groupes de malades. La radio-activité ventriculaire est mesurée par comptage externe 1 heure 3 heures et 24 heures après l'injection. Les valeurs sont exprimées en pour cent de la radio-activité mesurée à 1 heure.

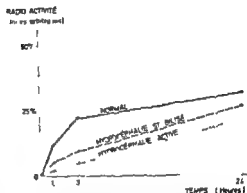


Fig 6 Courbes de la radio-activité sanguine dans les 3 groupes de malades. Les mesures sont faites par comptage externe devant l'aorte cardiaque. Les valeurs sont exprimées en pour cent de la radio-activité ventriculaire mesurée à 1 heure.

Dans les hydrocephalies dues à une obstruction ventriculaire l'indication d'une dérivation par ventriculo-cisternostomie peut se poser. Cette indication est fonction de la perméabilité des espaces sous-arachnoïdiens péri-cérébraux.

Lorsque ces espaces sont perméables, la radio-activité atteint les aires de résorption, mais avec un certain retard et il faut poursuivre l'exploration au delà de 24 heures (Fig 3). Dans ce cas l'encephalographie gazeuse avait montré une sténose de l'aqueduc de Sylvius. L'épreuve isotopique pré-opératoire était favorable à l'indication d'une ventriculo-cisternostomie, qui a été réalisée avec succès.

À l'inverse il existe dans certains cas d'obstruction ventriculaire des troubles associés de la perméabilité des espaces péri-cérébraux. La radio-activité reste alors bloquée à la base du crâne, comme sur la Fig 4 et il faudra recourir à une dérivation externe avec valve.

Tableau 1

*Evolution de la radioactivité ventriculaire après injection de serum albumine marqué  $\alpha$  à  $^{131}I$* 

État clinique	Nombre	Radioactivité ventriculaire en pour cent de la valeur mesurée à une heure	
		Trois heures	Vingt quatre heures
Normal	1	19 $\pm$ 10	5 $\pm$ 5
Hydrocéphalie stabilisée	9	80 $\pm$ 9	22 $\pm$ 11
Hydrocéphalie active	8	90 $\pm$ 1	15 $\pm$ 11

*Le contrôle post opératoire du fonctionnement et de l'efficacité des dérivations chirurgicales.* Ce contrôle est habituellement réalisé par injection ventriculaire de la serum albumine radio active et la mesure de l'évacuation du ICR ventriculaire et de ses échanges avec le sang.

À partir de 21 explorations nos résultats peuvent être répartis en 3 groupes (Fig 5, Tableau 1).

Un groupe normal où la décroissance de la radioactivité ventriculaire entre 1 h et 3 h est de l'ordre de 50 % et où la radioactivité ventriculaire résiduelle à la 24ème heure est inférieure à 10 %.

Un groupe où l'hydrocéphalie reste active. L'évacuation ventriculaire y est très lente: près de 10 % seulement entre 1 h et 3 h, la radioactivité résiduelle à 24 h est supérieure à 10 %.

Enfin un groupe intermédiaire où l'hydrocéphalie peut être considérée comme stabilisée. La décroissance de la radioactivité ventriculaire est de l'ordre de 20 % entre 1 h et 3 h et la radio-activité résiduelle à la 24ème heure est d'environ 20 %.

La Fig 6 et le Tableau 2 montrent l'écoulement de la radioactivité sanguine dans ces 3 groupes de malades.

*Contrôle des dérivations par ventriculo cisternostomie.* Les résultats des examens scintigraphiques faits après ventriculo cisternostomie peuvent être comparés à ceux d'un cas normal (Fig 7): celui d'un enfant de 9 ans où les investigations neuro-radiologiques (encéphalographie gazeuse et ventriculographie) avaient montré l'absence de toute anomalie du système ventriculaire et cisternal. Les scintigraphies montrent des ventricules de petite taille. Le liquide ventriculaire s'évacue de façon très rapide: à la 5ème heure, il en reste très peu.

Tableau 2

*Evolution de la radio-activité sanguine après injection ventriculaire de serum-albumine marquée à  $^{131}I$* 

État clinique	Nombre	Radio-activité sanguine en pour-cent de la radio-activité ventriculaire mesurée à une heure		
		Une heure	Trois heures	Vingt-quatre heures
Normal	4	$12 \pm 6$	$92 \pm 11$	$31 \pm 9$
Hydrocephalie stabilisée	7	$55 \pm 5$	$94 \pm 7$	$26 \pm 12$
Hydrocephalie active	6	$2 \pm 18$	$47 \pm 23$	$21 \pm 127$

dans les ventricules et la plus grande partie de la radio-activité se trouve dans les cisternes peri-cerebrales. A la 24<sup>ème</sup> heure, la radio-activité résiduelle imprègne les aires de resorption du LCR à la convexité du cerveau comme lorsque le produit est injecté par voie sous-arachnoïdienne.

L'exemple suivant (Fig. 8) est celui d'un contrôle effectué 3 mois après ventriculo-cisternostomie pour sténose de l'aqueduc de Sylvius. Cette intervention a eu un excellent résultat sur le plan clinique. La dynamique liquidienne est redevenue sensiblement normale. Les ventricules ont de petite taille et se vacuent rapidement par l'orifice opératoire dont la perméabilité est démontrée par ces images. Les espaces sous-arachnoïdiens sont perméables et le liquide radio-actif atteint les aires de resorption de la convexité d'une façon satisfaisante.

Dans l'exemple suivant (Fig. 9), l'exploration isotopique montre une stabilisation de l'hydrocephalie après ventriculo-cisternostomie. L'hydrocephalie était due à un blocage par tumeur du pulvinar. Cette tumeur se traduit sur les cisternographies sous forme d'une lacune située à la partie postérieure du 3<sup>ème</sup> ventricule. L'évacuation de la radio-activité ventriculaire est modérément ralentie, mais la circulation du LCR dans les cisternes périphériques vers les aires de resorption est satisfaisante.

Enfin à l'opposé des cas précédents la Fig. 10 illustre l'échec d'une dérivation ventriculo-cisternale effectuée pour sténose de l'aqueduc. Les ventricules restent très dilatés. Très peu de radio-activité sort par l'orifice chirurgical. L'évacuation ventriculaire est extrêmement lente et les ventricules restent encore imprégnés au bout de 4 jours. Ce contrôle post-opératoire devait inciter à réintervenir sans attendre pour mettre en place une dérivation ventriculo-cardiaque.

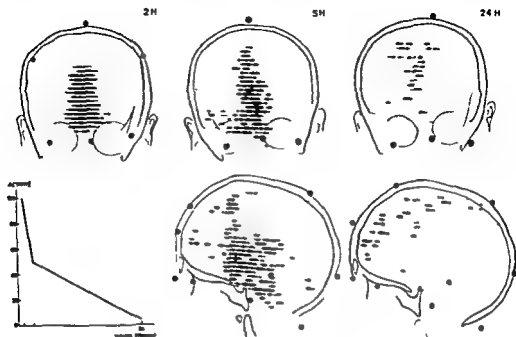


Fig. 7. Ventriculographie isotopique normale. Évacuation rapide de la radio-activité ventriculaire vers les aires de résorption de la convexité.

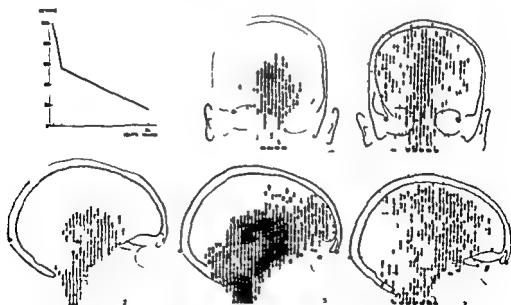


Fig. 8. Contrôle isotopique d'une dérivation ventriculo-cisternale pour sténose de l'aqueduc de Sylvius. L'intervention a eu un excellent résultat. Évacuation rapide de la radio-activité ventriculaire et régression satisfaisante des aires de résorption du LCR.

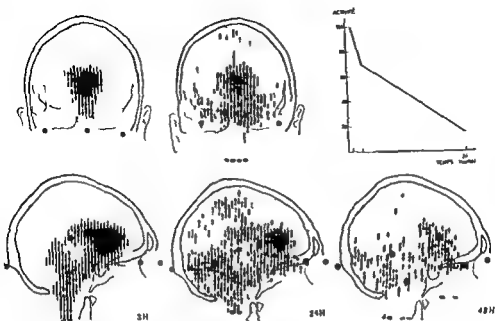


Fig 9 Contrôle d'une ventriculo-cisternostomie réalisée pour une hydrocéphalie par tumeur du puitsier. L'hydrocéphalie est stabilisée. Image lacunaire donnée par la tumeur à la partie postérieure du 3ème ventricule

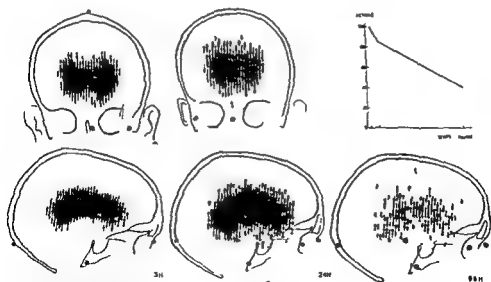


Fig 10 Contrôle d'une ventriculo-cisternostomie pour stenose de l'aqueduc. L'exploration scintigraphique confirme l'échec de l'intervention. Très faible évacuation de la radio-activité ventriculaire et absence de passage dans les externes péri-cérébrales

### Conclusion

La ventriculo-cisternostomie est une méthode simple et rapide de dérivation de certaines hydrocéphalies obstructives. Ses indications ont fondées sur la perméabilité des espaces sous-arachnoïdiens péri-cérébraux et sur une résorption physiologique satisfaisante du LCR.

La cisternographie isotopique pré-opératoire représente une excellente méthode pour tester cette perméabilité et les échanges physiologiques du LCR avec le sang.

Pendant le diagnostic d'obstruction ventriculaire a été fait bien souvent grâce à des explorations radiologiques (ventriculographie ou iodo-ventriculographie) qui obligent à intervenir sans délai. En raison de sa simplicité et de sa bénignité la ventriculo-cisternostomie peut être envisagée au décours même de l'exploration ventriculographique, quitte à recourir ensuite à d'autres dérivations si elle ne donne pas satisfaction. L'exploration isotopique post-opératoire intervient là encore de façon décisive pour apprécier les résultats fonctionnels de cette première intervention.

Les mesures de la vitesse de décroissance de la radio-activité du liquide ventriculaire peut servir sur le plan théorique à étudier le taux de renouvellement du LCR. Cette détermination nécessite cependant la connaissance du volume du LCR ventriculaire. Des études sont en cours pour mettre au point un mode d'appréciation de ce volume à partir des méthodes scintigraphiques.

### RÉSUMÉ

L'ouverture du plancher du 3ème ventricule dans la citerne interpedunculaire est un procédé de dérivation liquidienne applicable à certaines hydrocéphalies obstructives. Effectuée au trocart leucotomie sous contrôle radio-fluoroscopique cette ventriculo-cisternostomie est une intervention simple et bénigne. Son succès dépend de la perméabilité des espaces sous-arachnoïdiens péri-cérébraux. L'exploration par radio-isotopes fournit des informations morphologiques et dynamiques concernant le transit et la résorption du liquide céphalo-rachidien. Ces informations sont utilisées pour le choix de la technique de dérivation interne (ventriculo-cisternostomie) ou externe (ventriculo-atriale) et pour le contrôle de son efficacité. Après injection par voie ventriculaire la mesure de la décroissance radio-active permet d'étudier le renouvellement du LCR. Après dérivation chirurgicale interne ou par valve ces explorations servent à vérifier la perméabilité de la dérivation et l'efficacité de l'intervention.

### SUMMARY

Ventriculo-cisternostomy between the third ventricle and the interpeduncular cistern is a means of draining the cerebral fluid in certain cases of obstructive hydrocephalus. This is performed simply and safely by puncture leucotomy although its success depends on the permeability of the subarachnoid spaces. Radioisotopes furnish information on the morpho-

logy and dynamics of the reabsorption and passage of the cerebral spinal fluid information that aids in the selection of the method of drainage internal (ventriculo cisternal) or external (ventriculo atrial) and in its control. Furthermore the rate of decline in radioactivity will indicate the measure of renewal of the CSF. The introduction of an isotope serves to indicate the freedom of the drainage and the efficacy of the intervention.

## ZUSAMMENFASSUNG

Ventrikulocisternostomie zwischen dem dritten Ventrikel und der Cisterna interpeduncularis ermöglicht die Drainage der Gehirnflüssigkeit bei gewissen Fällen von obstruktivem Hydrocephalus. Diese kann einfach und sicher durch Punktionsleukotomie ausgeführt werden obwohl deren Erfolg von der Durchlässigkeit der Subarachnoidalräume abhängt. Radioisotopen verschaffen eine Information über die Morphologie und die Dynamik der Reabsorption und Passage der cerebrospinalen Flüssigkeit eine Information, welche bei der Auswahl der Methode für die Drainage behilflich ist der internen (ventrikulo-cisternalen) oder externen (ventrikulo atrialen) Methode und deren Kontrolle. Weiterhin bildet die Geschwindigkeit der Abnahme der Radioaktivität ein Mass für die Erneuerung der cerebrospinalen Flüssigkeit. Die Einführung eines Isotops dient als Indikator der freien Durchlässigkeit der Drainage und des Erfolges des Eingriffs.

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## FIVE YEARS EXPERIENCE WITH ISOTOPI CISTERNOGRAPHY AND VENTRICULOGRAPHY

by

G. J. ALKYS, JR., L. V. LESLIE and F. E. GLASAUER

Since first introduced by Di Chiro in 1961, isotope cisternography and ventriculography have gradually gained acceptance as a means of evaluation of the cerebrospinal fluid circulation. It seems appropriate at this time, to review and summarize our experiences with these examinations over the past five years and report our observations.

*Materials and Methods* A total of 110 examinations were performed on 106 patients. Our technique was described in detail in previous communications and will not be repeated here. For the purpose of this investigation, the patients were placed in six groups according to the suggested pathology for which the examination was performed.

*Cerebrospinal fluid leaks* A total of 111 isotope cisternograms were performed on 14 patients in this group, all with possible cerebrospinal fluid rhinorrhea, either post-traumatic or spontaneous in origin. There was no case of otorrhea in this group. In 7 patients the examination revealed cerebrospinal fluid leakage through the frontal or ethmoid sinuses into the nose. In 6 of these patients the findings were confirmed by surgery. The seventh patient has not

yet been operated upon. Two patients who had negative isotope cisternograms were found to have cerebro-spinal fluid rhinorrhea at surgery. One of these, an 11 year-old girl had an unusual source of leakage. She was found to have a congenital cleft in the sella turcica through which the dilated third ventricle herniated and leakage occurred through a defect in the floor of the ventricle. Since, normally, the radioactive material does not enter the ventricles leakage can only be demonstrated from the basal cisterns. This explains the failure to detect the radioactive material in the sinuses or nose of this patient. The second patient had a defect in the cribriform plate and there was no explanation for the failure to demonstrate it by isotope cisternography. In the case of five other patients surgery was not considered necessary in view of the negative cisternogram.

The only significant complications from isotope cisternography were encountered in this group. Two patients had a transient aseptic meningitis which cleared completely on symptomatic treatment alone. One of these patients was reported by NICOL (1967), the other has not been reported elsewhere. A third patient complained of severe headache and backache for 24 hours after the lumbar puncture without signs of meningitis.

In the evaluation of cerebro-spinal fluid rhinorrhea we rely on the findings of isotope cisternography. In fact in this group of patients no other tests were performed to detect the cerebro-spinal fluid rhinorrhea or localize its source.

*Normal pressure hydrocephalus.* The largest group in this series comprised those patients examined for possible normal pressure hydrocephalus. We included in this group those patients whose major presenting complaints were intellectual deterioration, ataxia and at times headache, and who on encephalography had normal cerebro-spinal fluid pressure and enlarged cerebral ventricles. Many of the patients were diagnosed as pre senile dementia and the examination was undertaken to differentiate between normal pressure hydrocephalus and cerebral atrophy. In our experience, and in that of others, patients with normal pressure hydrocephalus show improvement following ventriculo-atrial shunting. Patients with cerebral atrophy, however, fail to respond to surgical treatment. We considered the results of isotope cisternography to be the single, most important criterion on which to base the selection of patients suitable for ventriculo-atrial shunting.

Of the 44 patients included in this group 11 had onset of symptoms after subarachnoid hemorrhage or surgery. Almost all of the remaining 33 patients had some kind of head trauma preceding the onset of symptoms. In a few instances a clear history of trauma or hemorrhage could not be elicited.

The isotope cisternogram was considered abnormal if most or all of the

radioactive material injected into the lumbar subarachnoid space entered the cerebral ventricles with little if any spreading over the cerebral hemispheres. Twenty four of the 53 patients had abnormal isotope cisternograms. Of the 10 underwent ventriculo atrial shunting with varying degrees of improvement of neurologic deficits. In one patient of this group the improvement was really striking. This patient seen in December 1965, is to our knowledge, the first patient in whom the diagnosis of normal pressure hydrocephalus was made by isotope cisternography. He was the subject of a previous paper by us on normal pressure hydrocephalus. We re-examined him 15 years after surgery. His shunt is functioning well, his ventricles are nearly normal in size and his neurologic status is stable. In all patients in this category, isotope cisternography contributed to the evaluation of patients by establishing the cerebro spinal fluid flow pattern. The yield of patients suitable for shunting was less than anticipated. The less than striking results in those who underwent shunting is explained largely on the basis of underlying focal cerebral damage. Uncomplicated normal pressure hydrocephalus without focal brain damage is apparently uncommon.

*Compensated hydrocephalus in children.* All of our patients in this group were children referred from a birth defect clinic. All had larger than normal head circumference and varying degrees of neurologic deficits including a few with slight mental retardation. We undertook the evaluation of this group to establish the cerebro spinal fluid flow pattern in this entity and to see if any would likely benefit from ventriculo atrial shunting.

The isotope cisternogram was considered abnormal if the isotope wholly or partially flowed into the cerebral ventricles similar to that seen in normal pressure hydrocephalus. Clinically, the only significant difference between the two groups is the fact that in compensated hydrocephalus the patient's clinical status is stable over a relatively long period of time and the process is considered inactive.

Twenty four patients underwent a total of twenty six procedures including one isotope ventriculogram. Eight of these examinations were abnormal. The most interesting patients were a pair of identical twin brothers, aged 7, and their 17 year old sister, all of whom had clearly abnormal cisternograms. One of the twins had petit mal epilepsy, the other had no symptoms at all. Neither one had a history of previous significant head trauma or intracranial hemorrhage. The sister was somewhat mentally dull and had a history of meningitis in infancy. Her cephalogram showed slightly enlarged cerebral ventricles and normal filling of the subarachnoid space with air. We postulated that the underlying etiology in this family might be a congenital defect in the absorption of the cerebro spinal fluid by arachnoid villi. The mother, who had a somewhat larger than normal head, had a normal isotope cisternogram.

Another patient with an abnormal cerebro-spinal fluid flow pattern was three years post-operative for removal of an ependymal cyst of the third ventricle and was asymptomatic at the time of the examination.

In view of the stable clinical condition of the patients in this group none was subjected to ventriculo-atrial shunting. We no longer routinely investigate children for this purpose.

*Determination of shunt function* Fourteen patients who had previously undergone ventriculo-atrial or other shunts were examined to determine the patency of the shunt. This group underwent a total of 21 isotope examinations including 12 isotope ventriculograms. Isotope cisternography and ventriculography were found to be excellent means of determining the patency of the shunt. When the shunt is properly functioning the radioactive isotope injected into the lumbar subarachnoid space flows rapidly towards the shunt. Usually all of the injected radioactive material is in the ventricular system at 3 hours and at 24 hours most of it has already been removed from the ventricles and is in the circulating blood. The size of the cerebral ventricles can also be estimated by this method which is particularly useful since injection of air is considered by many to be hazardous in the presence of ventriculo-atrial shunts. Five of the 21 procedures in this group showed non functioning shunts. This was considered to be an indication for revising the shunt. All of these patients were re-examined post operatively and showed an increase in the speed of flow of the isotope towards the shunt and a decrease in the size of the ventricles.

We consider isotope cisternography and ventriculography to be the most valuable examination for determining the patency of shunts. Since these examinations are safe and relatively simple we do not hesitate to perform them if the question arises as to the proper functioning of the shunt.

*Hydrocephalus of miscellaneous etiology* A further group of 10 patients underwent isotope cisternography or ventriculography with the provisional diagnosis of hydrocephalus not fitting into any of the other groups outlined above. A total of 12 procedures were performed in this group including 7 isotope ventriculograms. There were two infants in this group with hydranencephaly in whom the diagnosis was established radiographically by injection of air along with the isotope. The isotope ventriculogram demonstrated a total lack of disposition of the radionuclide from the head. The drop of count rate over several days was essentially parallel to the physical decay of  $^{131}\text{I}$ .

Another patient in this group was a 4 month-old infant with obstructive hydrocephalus on the basis of meningitis as a newborn. He showed virtually no absorption of the isotope. The resolution of the Anger camera used for the isotope

ventriculogram was insufficient to show the grossly enlarged third and fourth ventricles demonstrated by ar ventriculography.

Ten of the twelve procedures performed in this group were positive. In these instances isotope cisternography and ventriculography were largely useful in demonstrating the lack of absorption or lack of flow of the cerebro spinal fluid. In this group, the limitations of isotope cisternography and ventriculography were also pointed out quite clearly since in none of the patients was anatomic detail of the underlying pathology demonstrable by the radionuclide method.

*Spinal lesions.* Finally, a group of six patients underwent a total of seven isotope cisternograms for investigation primarily of abnormality of the cerebro spinal fluid flow in the spinal canal rather than in the cranial cavity. Three of these had meningomyeloceles. In one patient with a large lumbar meningocele the isotope tended to pool in the meningocele both before and after partial repair of the meningocele. In two others the cisternograms were normal post operatively. Two patients in this group had cerebro-spinal fluid leaks, one through lower cervical nerve root avulsion demonstrated by myelography, the other post operatively after cervical spinal cord decompression. The leakage could not be demonstrated in either of these patients by this technique.

## SUMMARY

Isotope cisternography and ventriculography have been found to be simple and useful methods in the examination of selected patients with neurologic disease. The only significant complications were two cases of aseptic meningitis both in patients investigated for cerebro spinal fluid leaks. These examinations afford a method for evaluating the cerebro spinal fluid flow pattern which heretofore could not be satisfactorily demonstrated. The usefulness is mainly in the evaluation of patients with cerebro spinal fluid leaks, normal pressure hydrocephalus and patients in whom the patency of shunts is to be determined. We find these examinations less useful as screening tests for compensated hydrocephalus. Fine anatomic detail cannot be seen with the radionuclide methods.

## ZUSAMMENFASSUNG

Cisternographie und Ventriculographie mit Isotopen wurden als einfache und anwendbare Methoden befunden, um ausgewählte Patienten mit neurologischen Erkrankungen zu untersuchen. Die einzigen signifikanten Komplikationen waren zwei Fälle von aseptischer Meningitis, beide bei Patienten, die wegen cerebro spinalen Liquoristeln untersucht wurden. Diese Untersuchungen bieten eine Methode, die Strömungsverhältnisse des cerebro spinalen Liquors festzustellen, die bislang nicht zufriedenstellend festzustellen waren. Die spinalen Liquoristeln, Hydrocephalus mit normalem Druck und Patienten bei denen die Funktion eines Shunts geprüft werden sollte. Wir finden jedoch, dass diese Unter-

suntungen weniger brauchbar als Übersichtstests bei dem kompensierten Hydrocephalus sind. Feine anatomische Einzelheiten lassen sich nicht mit den Isotopenmethoden erkennen.

## RÉSUMÉ

La cisternographie et la ventriculographie isotopiques se sont révélées être des méthodes simples et utiles pour l'examen de malades sélectionnés atteints d'affection neurologique. Les seules complications importantes ont été deux cas de méningite aseptique toutes les deux chez des malades examinés pour rechercher des fistules de liquide céphalo-rachidien. Ces examens apportent une méthode de détermination du type d'écoulement du liquide céphalo-rachidien qui, jusqu'à maintenant ne pouvait pas être mis en évidence de façon satisfaisante. Ces examens sont particulièrement utiles pour étudier les malades atteints de fistules du liquide céphalo-rachidien d'hydrocéphalie à pression normale et de malades chez qui il faut étudier le fonctionnement des dérivations. Ces méthodes nous ont paru moins utiles comme épreuves de dépistage de l'hydrocéphalie compensée. Les méthodes radio-isotopiques ne permettent pas de voir de fins détails anatomiques.

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## CLINICAL ENCEPHALOGRAPHIC AND ISOTOPE INVESTIGATIONS OF HYDROCEPHALUS IN ADULTS

by

P AMUNDSEN, K KRISTIANSEN and J PRESTIUS

Sixty two adults were investigated during the period 1967 to 1969 for disturbances of the CSF circulation with a view to selecting those with communicating hydrocephalus for operation. The majority of the patients were between 40 and 69 years old and were mostly males (Table 1). The clinical diagnoses are given in Table 2. The group various intracranial lesions includes intracranial tumours, Huntington's chorea, Parkinson's disease and encephalitis, one patient had traumatic neurosis. All patients underwent isotope examinations of the CSF pathways and, except in 3 patients, encephalography was also performed. RISA was used for the isotope examinations, 200 to 400  $\mu$ Ci being injected into the lumbar subarachnoid space. Only one a.p. and one lateral scan was for technical reasons performed at 6 and 24 hours, 5 examinations were unsuccessful mainly due to restlessness in the patient (Table 3).

The intracranial activity was so low in 8 additional patients, either due to technical error or to a pathologic CSF flow, as to be of no value. One patient listed under special findings in Table 3 had a slow flow when the activity in the spinal canal was examined. Pneumography verified arachnoid adhesions. The remaining 4 patients in this group had local enlargement of the subarachnoid space. The interpretation of the scan is difficult under such conditions and two

**Table 1**  
*Age and sex distribution of the material*

Age	Males	Females	Total
<19	1	0	1 (16%)
20-39	4	2	6 (9.7%)
40-59	16	4	20 (30.3%)
60-69	18	10	28 (43.1%)
>70	0	2	2 (3.1%)
Total	44 (70.9%)	18 (29.1%)	62 (100%)

**Table 2**  
*Distribution of the material to clinical diagnostic groups*

Clinical diagnosis	Males	Females	Total
Head injuries	20	3	23
Subarachnoid hemorrhage	7	4	11
Encephalopathy of unknown etiology	11	7	18
Various intracranial lesions	6	4	10

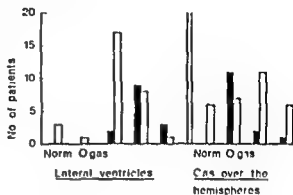
**Table 3**  
*Distribution of the material to isotope diagnosis*

Isotope cisternography	RIS 200-400 / Cs	
	Cases	Per cent
Normal	30	48.4
CSF obstruction	14	22.6
Special findings	5	8.1
Equivocal	8	12.9
Unsuccessful	5	8.1
Total	62	100

of these had been originally misinterpreted. Fourteen patients had activity within the ventricles and this was regarded as signs of abnormal CSF flow, a relatively high incidence as compared to similar materials from the literature.

The isotope examination in 30 patients was considered normal i.e. there was maximum activity over the cisterns at the 6 hours scan and activity over the hemispheres at the 24 hours scan when that over the cisterns had decreased. There was no ventricular activity. The correlation of isotope examinations and encephalography is evident from the Figure.





The size of the lateral ventricles and subarachnoid spaces over the hemisphere related to the CSF circulation determined by isotope examinations

The size of the lateral ventricles was larger in the group with an abnormal CSF circulation. None of the patients with intraventricular activity had normal sized ventricles while 3 patients in the negative isotope group had no enlargement of the lateral ventricles. Asymmetric enlargement of the ventricles was common in the normal but exceptional in the abnormal isotope group. 7 patients in the normal isotope group had pneumographic evidence of cerebellar atrophy. The subarachnoid spaces over the hemispheres were larger in the normal isotope group. These spaces are difficult to evaluate, it is however striking that 11 out of 14 patients in the abnormal isotope group had no gas filling over the hemispheres and in none were these spaces normal. The cisterns including the Sylvian cisterns, were wide in more than half the patients although there was no difference between the normal and abnormal isotope groups.

The distribution of patients with normal and abnormal CSF circulations had no preference for the different clinical groups (Table 1).

Ventriculo atrial shunt ad modum Pudenz Heyer was performed in 10 patients all males. 5 of these improved (one aged 39 with brain contusion, one aged 52 with intracerebral hematoma, one aged 37 with subarachnoid hemorrhage, one aged 66 with intracerebral cyst, and one aged 67 with cerebral metastasis).

A few comments on the patients who failed to benefit from the intervention will be made. The first patient, aged 64, had severe brain contusion and gradual increase of ventricular size and cisternal spaces at repeat pneumography. The next patient, aged 18, had focal contusion of the left temporal lobe that resulted in cystic dilatation of the subarachnoid space. The isotope collected and was wrongly interpreted as intraventricular activity. The third patient, aged 67, was diagnosed as having Alzheimer's disease at the post mortem examination. The pneumographic appearances were however peculiar since there was marked dilata-

Table 4

*Correlation between clinical groups and CSF circulation determined by isotope examinations*

Clinical group	Abnormal CSF flow	Normal CSF flow
Encephalopathy	4	13
Subarachnoid hemorrhage	3	6
Brain contusion	5	13
Various	2	6
Total	14	38

tation of the lateral ventricles and practically no gas over the hemispheres. The isotope examination revealed low intracranial activity, originally interpreted as indicating reflux to the ventricles, this was probably wrong.

One of the two patients (aged 51 and 56, respectively) with a clinical diagnosis of encephalopathy of unknown etiology had gas as well as isotope activity over the hemispheres. A shunt in such instances is probably of little help to the patient. No explanation is forthcoming as to why the last patient failed to benefit from the shunt.

## SUMMARY

Encephalography and isotope examinations of the CSF circulation were performed in 62 adults. The findings were correlated with the symptoms and clinical signs. A ventriculo-atrial shunt (Pudenz Heyer) was performed in 11 patients.

## ZUSAMMENFASSUNG

Encephalographie und Untersuchungen mit Isotopen der Zerebrospinalflüssigkeitszirkulation wurden bei 62 Erwachsenen durchgeführt. Die Befunde wurden mit den Symptomen und klinischen Zeichen korreliert. In 11 Patienten wurde ein ventrikulo-atrialer Shunt (Pudenz Heyer) vorgenommen.

## RÉSUMÉ

Les auteurs ont fait chez 62 adultes des encephalographies et des examens isotopiques de la circulation du liquide cephalo-rachidien. Les resultats ont ete confronte avec les symptomes et les signes cliniques. Une derivation ventriculo-atriale (Pudenz Heyer) a ete fait dans 11 cas.

## FURTHER INVESTIGATIONS ON PULSATILE MOVEMENTS IN THE CEREBROSPINAL FLUID PATHWAYS

by

G. DU BOULAY, J. O'CONNELL, J. CURRIE, THEA BOSTICK and PAMELA VERITY

In 1966 some radiologic observations of pulsatile movements in the cerebrospinal fluid pathways were described by du Boulay who was working upon O'Connell's idea of a CSF pump. Now, as a result of further work, the earlier conclusions have been modified and widened. The present findings throw light upon the CSF circulation and possibly, as will be reported separately, upon the aetiology of hydromyelia, some forms of hydrocephalus and of arachnoid cysts. The work to be described here is intended to put the whole subject into perspective.

A brief review of the literature may be helpful. Leaving aside early observations establishing the existence of pulsatile pressure changes, most published work is concerned with the following main topics: (1) If the source of the pulsatile pressure waves coincident with heart rate is (a) arterial in the head, (b) arterial in the spine, or (c) venous. (2) Granted that a large part of the pulse pressure wave derives from the head, the question arises if it comes from (a) expansion of arteries at the base, (b) expansion of the vessels within the brain, or (c) expansion of the choroid plexuses. (3) The means by which the respiratory fluctuation of CSF pressure is brought about. (4) The relationship of alterations in cerebral blood flow to CSF pressure changes. (There are also

many observations upon abnormal conditions, notably increased CSF pressure, which will not be dealt with here.)

For obvious reasons much of the work is experimental upon animals, and as is usual with such work there is a tendency to regard experimental results on the dog as valid for monkey and man. Even cats have been employed. It is however, necessary to remember always which mammal is under discussion. There are fundamental differences in their arterial and venous anatomy which may influence the pressure waves.

There is very little work upon the pulsatile movements which result from pressure changes. The sources of these pulsatile pressure changes coincident with the heart rate were dealt with by ZIEGLER (1896) in dogs, BECHER (1922) in dogs and man, MYERSON & LOWAN (1932), ANTONI (1933) and O'CONNELL (1943) in man, BERING & INGRAHAM (1953) and BERING (1955, 1962) in dogs, SCHILD *et coll* (1956) in humans, BENDER (1963) and GUTHRIE *et coll* (1966) in man, GROTE (1964), DUNBAR *et coll* (1966) and ADOLPHE *et coll* (1967) in dogs.

The majority of workers throughout these seven decades have become convinced that the cardiac CSF pressure rise measured in the ventricles at the cisterna magna and in the lumbar theca, is caused by the rhythmic arterial input of blood to the cranial cavity. Their conclusions are based upon (1) Timing in relation to the carotid pulses and ECG and various venous pulses and heart sounds. BECHER (1922) in dogs and man, SCHILD *et coll* (1956) in man, SALATHE (1876) and BENDER (1965) in man. (2) Upon the character of the pulse wave, ZIEGLER (1896) in dogs, BECHER (1922) in dogs and man, BENDER (1963) in man, DUNBAR *et coll* (1966) in man. (3) Upon its alteration by obstruction elsewhere in the vascular system. FALKENHEIM & NAUNY (1887) right ventricle in dogs, DUNBAR *et coll* (1966) aorta in dogs.

A very few have suggested as a result of their experiments that the cardiac CSF pulse has venous rather than arterial characteristics. FALKENHEIM & NAUNY (1887), ADOLPHE *et coll* (1967), HAMIT *et coll* (1965), however point out that the pulse waves recorded may, under differing circumstances, show the features of both arterial and venous sources, a conclusion which common sense must acknowledge as the most likely. The other observers may well have been misled by the insensitivity of their recording gear.

Much of the recent discussion about the method whereby arterial pulsations are imparted to the CSF within the head stems from BERING's work (1955, 1962), DUNBAR and his co-workers' complex animal experiments (1966) and WILSON & BERTAN (1967).

Most authors e.g. BECHER (1922) had envisaged the arterial inflow to the head as causing an expansion of the brain and of vessels within the basal

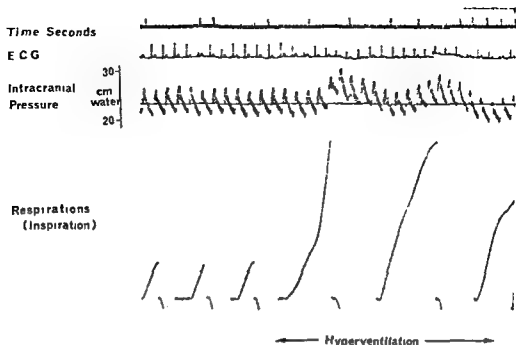


Fig 1 Human subject. Time relationship of the CSF pressure rise and in piration is different in quiet respiration and in forced hyperventilation (From BERTILS *et coll* (1968))

cisterns O'CONNELL (1943) suggested that the brains expansion by compressing the third ventricle might constitute a CSF pump. BERING believed as a result of his experiments that the thrust imparted to the CSF came from choroid plexus expansion and GARDNER (1965) has accepted this idea but there has never been any direct evidence of choroid plexus pulsation and simple physical principles make it doubtful whether it can contribute a major share to CSF movements or to the pressure wave.

The observations of FALKENHEIM & NAUNYN (1887) of KNOX (1886) and of BECHER (1922) — in dogs for the most part — drew attention to the effects of respiration upon CSF pressure. They were able to show that in spontaneous respiration a rise of CSF pressure at the cisterna magna took place in expiration but controlled respiration on curarised animals reversed this state of affairs and there was then an inspiratory rise. KNOX provided some evidence that it was the spinal veins which were in part responsible by transmitting thoracic and abdominal pressures.

The importance of the spinal veins as a cause of alterations of CSF pressure has often been overlooked though re-emphasised from time to time. CURRIE'S

investigations of intra ventricular pressure in humans (BETHUNE et coll 1968), have drawn attention particularly to the variations of phase of the respiratory pressure rise between inspiration and expiration depending upon posture and the type of respiration (Fig 1)

The inter relationship of changes in cerebral blood flow and CSF pressure is of current interest HEDGES & WEINSTEIN (1964) worked with monkeys to determine the effect upon carotid arterial and superior sagittal sinus pressures of artificially raising the CSF tension

CUSHING's classical experimental observations on the effects of a CSF pressure rise diminishing cerebral blood were confirmed in man by KETY et coll in 1948

Working on the opposite aspect of the phenomena, RYDER et coll (1951 1952) using rhesus monkeys, demonstrated that changes in cerebral blood flow affected the CSF pressure No great headway has yet been made in defining the inter relationships of cerebral blood flow and CSF pulse pressures

*Material* Occasional observations were made upon patients undergoing positive contrast ventriculography before publication of the previous paper (DU BOULAY 1956) and these are now added to the present material which consists of (1) Measurements made during encephalography under TV control on a further 93 patients (2) Measurements made similarly at 7 positive contrast ventriculographies (3) Measurements made at 3 air ventriculographies (4) Measurements made from video tape records of a further 87 positive contrast myelographies (5) Some investigations carried out on intra cranial and cisternal pressures by one of us (J C) (6) Experimental observations of CSF movements and pressure in 6 rabbits 2 cats, 2 goats and 6 Patas monkeys (7) Post mortem injection examinations of the cranial arterial system of 110 species of mammal (P V) (8) A detailed investigation of caudal vena caval pressure and flow in a variety of animals has also been carried out by FIEVES DU BOULAY GABRIEL & VERITY (unpublished) and observations upon some of these are relevant to this work (17 rabbits, 2 cats 1 coyote, 1 timber wolf, 3 goats and 6 Patas monkeys)

Measurements of fluid levels and their movement at encephalography have been made directly upon the face of the TV monitor with a transparent ruler and subsequently corrected for magnification from films taken as nearly simultaneously as possible By this means the lengths of fluid levels are likely to be accurately known but no method has been devised for testing the accuracy of the measurements of amplitude

Myelographic examinations have been recorded upon a Sony video tape recorder The inherent difficulties of measurement in these are not quite the same because a movement may be replayed as often as necessary

In the animal investigations greater accuracy has probably been achieved by using telecine recording (Marconi) and measuring movements of the image of the positive contrast medium by means of a Vanguard analyser. It has been shown experimentally that variations in the diameters of small amounts of positive contrast medium measured by this system on 16 mm telecine film are significant when greater than 0.18 mm. In fact pulsatile movements have only been accepted when easily visible to the naked eye, i.e. of the order of 1.0 mm.

In all cases Marconi television systems have been employed (either 4 1/2" image orthocon or icon) and for the pneumographies the television chain has been at low gain to obtain maximum resolution.

Pressure measurements have been made in two different ways. In some humans a pressure transducer attached to an indwelling ventricular drain has been radio-telemetered so that changes in pressure with posture may be more easily recorded.

In other cases a Statham strain gauge has been connected directly by pressure line to a cisternal needle.

In the animals a Statham D23 BB gauge was similarly connected to ventricular or cisternal needle without loss of CSF and the trace recorded by cine photography of an oscilloscope face.

Observations have been directed towards the following problems: (1) If the sources of pulsatile movement of CSF as described in 1966, i.e. (a) systolic brain expansion and (b) arterial expansion are to be found in the basal cisterns; (2) If there is any supporting evidence for BERING's suggestion that choroid plexus expansion is a source of CSF pulsation; (3) If there are any movements not previously taken into account; (4) Why the observed movements vary in their amplitude; (5) The physiologic significance of the movements. (The question what alterations take place in pathologic states particularly in Chiari malformations and in the presence of cerebral tumours has also occupied our attention but is not dealt with here.)

*Discussion on the timing of movements.* In order to determine the cause of a pulsatile movement one would ideally time the relationship of each movement against the systolic peak of pressure in each artery or vein possibly initiating that movement. Such a simple scheme of investigation is extremely difficult to apply to the movements of CSF because in most cases the points at which CSF movements may be most easily observed are at considerable distances from the probable causes of the movement and from the point where a pressure wave may be recorded. Pulsatile movements take a finite time to be propagated. They pass through channels having complicated anatomy and there is a variety of possible sources of pulsation along their pathways. So, for example, neither a record of the carotid pulse nor an ECG tracing is of overwhelming importance

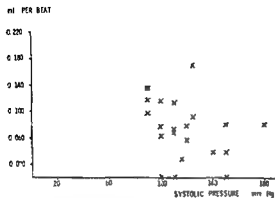


Fig 2 Volumetric displacement versus arterial systolic pressure in third ventricle. Little correlation between systolic pressure and the volumetric CSF displacement.

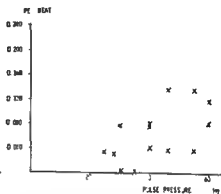


Fig 3 Volumetric displacement versus arterial pulse pressure in third ventricle. Relationship between the pulse pressure and volumetric CSF displacement.

in determining the source of movement observed in the dorsal spinal canal during myelography. They may be useful in other ways.

A second method of determining the cause of movement in a system open to atmospheric pressure is to interrupt the pathway. If a movement beyond the interruption stops, the source may again be presumed. Here again is a method which has some application in working out the causes of CSF pulsation when, for instance, an intermittent block in the cervical canal may be brought into operation by extension of the head, thus cutting off any cerebral pulsation from exerting its effects upon the dorsal spinal canal. There are, however, difficulties in interpreting the results, for the CSF lies in a space which is only vented to atmospheric pressure through the venous network, and such elasticity as exists must reside in the veins. Thus, on theoretical grounds, pulsatile movement may be abolished distal to a block in the spinal canal by cutting off the source but also proximal to the block by denying any access to the area where elasticity lies. A further difficulty is the probable multiplicity of sources of pulsation.

For all these reasons, the simultaneous recording of arterial pressures or ECG traces with recordings of CSF movement, though used in this work, has formed only a part of the basis for estimating the origins of pulse waves.

There is another means by which the direction of propagation of a CSF pulse wave may be determined; this is by paying attention to the nature of the to and fro movement which contrast medium undergoes when the CSF pulsates. The systolic thrust moves the contrast medium rapidly in the direction of



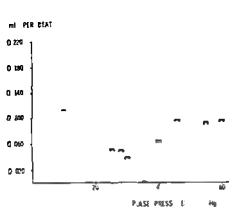


Fig 4 Volumetric displacement versus arterial pulse pressure in third ventricle (mean values). Single observations (straight lines) are given at some pulse pressures in the central part of the graph; the low mean value for CSF volumetric displacement includes several zero observations.

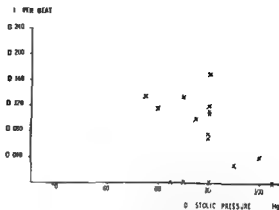
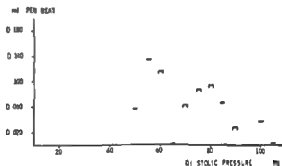


Fig 5 Volumetric displacement versus arterial diastolic blood pressure in third ventricle. Inverse relationship between the diastolic blood pressure and volumetric CSF displacement.

Fig 6 Volumetric displacement versus arterial diastolic blood pressure in third ventricle. Mean values.



propagation of the wave. It returns to its former position more slowly in diastole, and if the heart rate is reasonably slow, say slower than 90 beats per minute, the diastolic pause is evident. These considerations apply when the contrast medium is neither falling nor rising under the influence of gravity. The direction of systolic thrust may also be seen during the passage of myodil under gravity's influence as a subject is tilted: for when it falls towards the source of the pulse wave, the fall is step-like: rapid in diastole, arrested or even reversed in systole for a brief moment. These methods have proved to be a potent source of information and are considered to be highly reliable in determining the direction of pulse wave propagation.

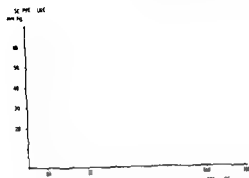


Fig 7 Systolic blood pressure versus pulse pressure. These are directly proportionate.

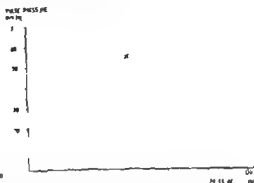


Fig 8 Arterial diastolic pressure versus arterial pulse pressure. These are not proportionate.

### Observations in man and their interpretation

*The third ventricle* When a fluid level lies entirely behind the massa intermedia at encephalography a figure for the volumetric displacement of fluid in the third ventricle may be calculated from (1) the amplitude of up and down movement of the fluid level (2) the length and (3) the measured width of the third ventricle on other films. One may assume an approximately rectangular figure enclosed by these measurements.

An attempt has been made to discover (1) the average quantitative volumetric CSF displacement through the third ventricle in normal cases (it is  $0.069 \pm 0.026$  ml ranging from 0 to 0.135 ml) and (2) how the volumetric displacement varies with the arterial blood pressure. The figures given have been corrected for optical enlargement.

The graphs appear to show that (1) Volumetric displacement is not in a simple way related to the systolic blood pressure. If anything there is a tendency for larger pulsatile displacement at low systolic pressures (Fig 2). (2) Larger volumetric displacements occur at high and low pulse pressure than in the middle range (Figs 3-4). (3) Low diastolic pressure is associated with larger volumetric displacement (Figs 5-6). (4) Systolic blood pressure and pulse pressure rise together but there is no close relationship between diastolic pressure and pulse pressure (Figs 7-8).

Readings from two of the patients in whom the pressures varied during a single examination illustrate some of these phenomena.

*Case 1* A patient who suffered from visual disturbance due to a pituitary tumour. At the beginning of the examination her blood pressure was 130/100 and the air/CSF fluid level was almost immobile. Very soon after this air in the third ventricle suddenly began to show a pulse so marked that the anterior half of the third ventricle seemed to empty com-

pletely being squeezed quite flat during systole and coincidentally the patient felt faint presumably with a very low blood pressure. Within a minute or two she felt better, the blood pressure having again risen and settled at 90/70 and the CSI pulse was again very slight.

**Case 2.** A suggested parietal tumour which could not be verified. Mild hypertension (BP varied between 150/100 and 180/120). With the patient sitting, there was a marked pulse in the third ventricle (corrected volumetric displacement 0.08 ml, observed amplitude  $> 2$  mm). Simultaneously a fluid level present in the basal cisterns showed no movement (an unusual finding, but one which clearly demonstrated the fact that the third ventricle pulse was not dependent upon the direct effect of expansion of basal cistern nor upon the movements of basal arteries).

BETHUNE *et coll.* (1968) and others have shown that a high arterial systolic pressure is generally accompanied by a high CSI pulse pressure. The dissociation shown here between high arterial pressure and volumetric pulsatile CSI displacement in the third ventricle may perhaps be explained by the dependence of the pulsatile volumetric displacement upon the turgidity of the venous bed which in turn is dependent upon the central venous pressure.

KNUDSEN (1958) gives the average volume of the third and fourth ventricles combined as 1.5 ml and the average volume of the third ventricle, aqueduct and fourth ventricle in EAST & LOMSTERS (1953) 21 specimens was 1.19 ml. The third ventricle may be estimated to make up about half of this volume, that is 0.5 to 0.75 ml. The displacement of 0.1 ml of fluid would be accomplished in a third ventricle of 7 mm diameter by 1 mm approximation of the walls — a movement difficult to observe radiologically. In cases of atrophy, however, a greater movement of the ventricular wall may be observed as in one such case where the volumetric displacement amounted to 0.176 ml.

It will be shown later that there is little correspondence at the cisterna magna between CSI displacement and blood pressure. It is therefore of interest to speculate why some correspondence may be observed between the CSI displacement in the third ventricle and both the arterial diastolic pressure and the arterial pulse pressure.

The third ventricle is much narrower than the cisterna magna and therefore may be regarded as a more sensitive instrument for measuring alterations in volume. In addition the volumetric displacement of CSI in the third ventricle represents only part of the total effect of brain pulsation. The pump action in the third ventricle might be expected to become a prominent feature when (1) outward expansion of the brain towards the vault is limited because elasticity available from CSI displacement out of cortical spaces has been used up, the cortical layer of CSI having become thinned out and (2) the available elasticity of the venous bed in the brain itself is reduced because low venous pressure has allowed the veins to collapse.

It is, therefore, not surprising to find that volumetric displacement through the third ventricle increases both as the arterial pulse pressure rises above normal and also in hypotension.

Watching myodil passing through and poised against the edge of the foramen of Monro one becomes convinced, (DU BOULAY 1966) that CSF does not travel back into the lateral ventricle. The fluid at the foramen of Monro is still, nor is the front of the third ventricle expanded during systole, rather the reverse may happen. Fluid does, however, travel down the aqueduct immediately following the third ventricular squeeze and if there is no obstruction fluid at once passes out of the foramen of Magendie. Thus, about one fifth of the third ventricle fluid is forced down the aqueduct with each heart beat and an unknown quantity, visually estimated to be about the same, leaves the fourth ventricle. Immediately afterwards during diastole most of this will return. These to and fro movements must be fairly effective as a mixing device between the small ventricles and the cisterna magna.

*The basal cisterns.* During many encephalographies but for only a very short time with the patient sitting during the filling phase a bubble of air becomes trapped in the interpeduncular cistern and upper part of the pontine cistern while fluid still remains in the medullary cistern and the lower part of the pontine cistern. A fluid level is seen at the bottom of the bubble in front of the pons and this fluid level almost invariably moves downwards along the clivus during or soon after each systolic inrush of blood to the head. The bubble is usually rather narrow from side to side rarely extending the width of the pons. Its movements therefore represent the movement of only a narrow column, only a small proportion of the whole amount of fluid surrounding the brain stem. Calculation however based upon the size of the bubble shows that the displaced volume of even this small proportion is on average as great as the displaced volume of the third ventricular fluid with each heart beat. The total volume of the CSF in the basal cisterns is at least ten times as great as the volume of the bubble.

Myodil examinations show that all the basal cisterns CSF moves. Thus the displacement of CSF per heart beat in the basal cisterns is much greater than the displacement in the third ventricle. There can be little doubt that it is the expulsion of basal cistern fluid into the spinal canal rather than of ventricular CSF which is mainly responsible for the spinal CSF pulsatile movement in the upper cervical canal. Considerable movements have also recently been observed during the passage of positive contrast medium through the cerebellopontine angle cisterns.

Two alternatives commend themselves for the investigation on the causes of movement in the basal cisterns. Firstly, here lie the main arteries of the circle

of Willis their systolic expansion may be the reason for fluid displacement. Secondly brain expansion alone must drive out fluid from over the convoluted surface of the cerebrum: it can only move towards regions able to expand to receive it. The mechanics of the parasagittal region require further investigation, and whether fluid is forced towards the brain's upper surface in systole remains unproven. It does seem however, most probable that fluid from the lower sulci must be forced downwards towards the skull base.

The part played by arterial expansion in the basal cisterns is susceptible both to observation and calculation. A common observation is the up-thrust of the terminal carotid and its branches into the air above the sella in the lateral view — always in systole. Less often one may see very small movements of the basilar artery or of the vertebral arteries during myelography. On two occasions only has a movement of the anterior part of the floor of the third ventricle been observed at a point where it might be explained by systolic elongation of the basilar artery and it remains a possibility that this particular movement — a systolic indentation just posterior to the infundibular recess in cases with post-fixed optic chiasms — was only the result of inequality of pressure between cisterns and ventricle. At all events it is a very small and very uncommon movement in normal cases.

Although such movements as these are seen the principal impression is of a rather inflexible arterial system. Pulsatile expansion of the vessels is not obvious. Although it must occur and can be seen at operation the increase in diameter of the vessels in systole must be small.

One may calculate within broad limits the increase in diameter which would be necessary in arteries at the brain's base if the brain alone were to be responsible for the occasionally observed expulsion of 3 ml of CSF from the skull during systole.

Internal diameters of the supracavernous carotid, precommunicating anterior cerebral, main trunk of posterior cerebral and posterior communicating vertebral and basilar arteries have been measured upon angiograms under known conditions of  $\text{paco}_2$  and blood pressure. Some of these measurements are published in du Boulay et coll. (1968, 1970). It is assumed that the diameters measured lie between the extremes of systole and diastole. The arterial diameters have been measured at both the lower and upper limits of normal  $\text{paco}_2$ . Taking the mean diameters one may calculate an average volume of the basal arterial system to be in the region of 0.8 ml.

In order to add 3 ml to its volume that is approximately to quadruple it the mean diameter would have to be doubled. Even the displacement of 1 ml of CSF from the basal cisterns by arterial expansion would imply a doubling of arterial volume and the addition of 1 ml to the diameter of a 2 mm vessel.

It is an observed fact at operation that nothing like this degree of expansion takes place. The conclusion to be drawn is that fluid is forced downwards through the basal cisterns during systole largely by brain expansion.

It is unfortunate that up to this time the superficial subarachnoid space over the brain has not proved easy to examine radiologically in any way which might reveal motion. Cisterna ambiens on the other hand is observable.

*Cisterna ambiens.* Observations were made of cisterna ambiens from time to time when good lateral views of it were obtained. Attention was particularly directed to it in encephalograms in the sitting position from No. 64 onwards.

Pulsatile alterations in size or shape of cisterna ambiens were seen in three cases for certain and in two with some uncertainty.

*Case 3.* Cisterna ambiens was seen to become narrower at the same time as the fluid level in cisterna magna dropped. In this case at this time the BP was 140/80 mm Hg (having risen to this figure from 120/70). There was simultaneously a pulsating fluid level in the cisterna magna 2-3 mm amplitude  $\times$  12 mm length. The downward thrust in the basal cisterns followed a detectable interval after the radial pulse but it was systolic in type. Pulse rate at the time 70.

*Case 4.* BP 110/80 mm Hg having risen from 100/70 with cisterna magna pulsating fluid level 2 mm amplitude  $\times$  15 mm length and pontine cistern movement anterior to the basilar artery 5 mm amplitude  $\times$  2 mm length. Cisterna ambiens became narrower as the fluid level in cisterna magna dropped. In this case downward movements of cisterna magna and pontine cistern coincided with the radial pulse.

*Case 5.* Cisterna ambiens pulsed getting lighter in colour on the television monitor (i.e. presumably wider) as the cervical fluid level moved downwards. Timed against the (visible) carotid termination the beginning of cisterna magna downward movement was perhaps just before systole in the head. In this case as usual the pre-pontine fluid level went down as the carotid moved up i.e. in systole.

Observations in the first two cases are consistent with general brain expansion during its systolic filling with arterial blood. In the last case some other explanation should be sought, maybe unequal expansion of different parts of the brain caused part of the cisterna to gape. Alternatively the downward movement of the fluid level in the cisterna magna might have been partly dictated by spinal venous collapse, causing movement to begin before the carotid pulse wave reached the brain.

In other cases the air-filled cisterna ambiens moved apparently with respiration probably opening up in inspiration.

### Spinal canal

A careful watch was kept upon the effects of respiration of coughing and of straining upon the movements of myodil in the spinal canal during myelography.

*Coughing* Twelve patients, without tumour or other CSF obstructions in the spinal canal were asked to cough while lying prone, horizontal and with various degrees of tilt feet down up to standing position. The myodil lay in the lower lumbar sac.

Coughing was usually preceded by a short sharp inspiratory effort sometimes more obvious than at others followed by an explosive expiration against the partly closed glottis. The upper level of contrast medium in the spinal canal, with the patient semi erect dropped sharply at the very beginning as the patient drew breath. This drop was followed by a sudden and definite rise of the upper level of the contrast column coinciding with the expiratory effort the contrast medium sometimes moving upwards as much as the width of two whole vertebral bodies but more often the movement was of only about 1 cm. This upward rise resulted from and was accompanied by a narrowing of the spinal theca sometimes as much as 50% as seen in a p.p. projection. Thus it was clear that the theca was being compressed during coughing by the tissues around it and only the epidural veins are capable of such an action. In only 2 out of the 12 cases was there no upward movement. The amount of CSF displaced upwards within the lumbar spinal canal may within wide limits be calculated from the amplitude of the movement and the estimated size of the spinal canal. If nerve roots occupy three-quarters of the subarachnoid space the volume of fluid displaced upwards across a fixed point by a cough would usually be between 2 and 9 ml.

Thus it is clear that such a free and effective pathway exists for the transmission of abdominal pressure to the caudal theca that CSF in considerable quantities is displaced in a cranial direction. The question then arises in what structures the elasticity resides which accommodates this displacement.

Myodil may be observed in the cervical region with the patient horizontal or head down. Here also a cough propels the myodil in a cranial direction some of it actually into the head from whence it immediately returns after the cough is finished. Elasticity must therefore lie within the head. In some patients the myodil may be propelled upwards as much as 4 cervical vertebral segments which must represent an injection of between 4 and 8 ml of CSF into the head.

It is surprising that pressure transmission in this long pathway from abdomen, via abdominal vena caval tributaries to epidural veins and then up the whole length of the spinal canal can be sufficiently forcible to overcome the intracranial pressure more or less simultaneously transmitted from the thorax via the right atrium and superior vena cava along a shorter pathway to the cerebral veins. The thecal pathway however probably receives a reinforcement of compression from thoracic and cervical veins and that there is an asynchronous arrival of the two shock waves in the head is shown by the rapid oscillation of

the myodil across the foramen magnum. Within the head only the vessels exist to accommodate an increase in CSF volume. The brain tissue itself is incompressible by such sudden shocks.

At all events a cough must compress the cerebral vessels that circulation of blood is for a short while greatly retarded and may even cease.

*The valsalva manoeuvre.* A similar phenomenon may actually be observed at angiography during a valsalva manoeuvre. Contrast injected into one of the main arteries in the neck will often reflux down another — for instance, from one vertebral artery to another. This same valsalva manoeuvre if carried out forcibly may also be shown at myelography to compress the lower spinal theca, displacing the CSF in a cranial direction and some actually into the head. During such a prolonged effort there is no possibility of a rapid oscillation of CSF and the cerebral circulation must accommodate itself or cease. (The further pursuit of such reflections is outside the scope of this paper.)

In some animals the cardiac pulsatile pressure variation of the posterior vena cava is greater than the respiratory. Measurements of the human inferior vena caval pressure (FRANKLIN 1937) suggest that there is a wide range of normal responses and that sometimes a significantly pulsatile pressure exists. A free pathway for respiratory pressure transmission to the spinal CSF has been shown and it seems reasonable to expect a vascular cardiac pressure wave also to be transmitted via epidural veins.

In the great majority of cases however, as described in 1966 the pulsatile impulse of the CSF is downwards towards and into the lumbar theca. Greater attention has however, been paid to pulsatile movements in the spinal theca and new features have occasionally been observed.

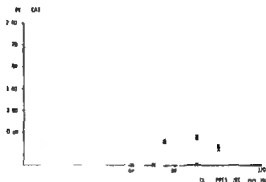
Out of the material of 87 myelographies 5 were seen to have systolic type of pulsation in a cranial direction in the lumbar (2) or dorsal (3) regions and in 4 of these the direction of the pulse could be reversed by various manoeuvres. Usually merely altering the angle of tilt by a few degrees was sufficient.

All three patients who showed a cranially directed systolic pulse in the dorsal region were abnormal. Two had compressive dorsal arachnoid diverticula and one a severe cervical obstruction due to spondylosis. It seems likely that the partial obstructions acted as valvular mechanisms tending to cut off the cranial pulse but permit the smaller lower spinal (venous) pulse to pass.

The only obvious explanation of these phenomena is that a pulsatile driving force must also exist in the spinal canal and that a substantial part of this force lies in the cauda equina region. The arteries of the region seem insufficient to provide so much rhythmic variation of volume and it is much more likely that the epidural veins are the origin of the pulse. Variation of the direction of the pulse with posture in the lumbar region may easily be explained because the



Fig. 9 Volumetric displacement versus arterial diastolic pressure in cisterna magna. Arterial diastolic pressure and arterial pulse pressure are not proportionate



inferior vena caval pressure is posture dependent and the turgidity of spinal veins must also be directly related to posture.

The momentary rises in pressure in spinal veins and in cranial arteries initiate CSF pulses in opposition. The direction in which CSF is driven at any particular point along the spinal canal must depend upon the relationship of the two opposing pressure rises both in time and amplitude. Since moreover, the venous system is responsible for providing elasticity for CSF movement, the maximum amplitude of arterially caused movement must be related to the volume of the veins and their compressibility.

To take a theoretical example. If for some reason the spinal veins were largely collapsed but filled momentarily as a result of a peak of IVC pressure the conditions would promote a CSF pulse in a cranial direction. This would be even greater if the pressure wave initiated in the head reached the lumbar region at a moment when the spinal veins were in a collapsed state for the absence of the elasticity afforded by them when full would prevent the possibility of CSF movement towards them. Even a high cranially originated pressure wave would fail to cause CSF movement. The net result might be that a low abdominal venous pressure coupled with a high intracranial arterial pressure resulted in CSF pulse movements directed towards the head — a state of affairs which would explain exactly the observations made when some patients are tilted head downwards.

*The cisterna magna.* For the purposes of easy nomenclature 'cisterna magna' is taken to include the wide spaces behind the cervical cord at C1. It is here that the movement of fluid levels is large and obvious during encephalography in the sitting position.

The cisterna magna, lying at the junction of ventricular, extra cerebral and spinal CSF pathways is subject to the forces created in each. The complex movements in normal cases are of great interest.

In 27 cases it was possible to measure the cross-sectional area of the space enclosed by the posterior arch of the atlas by taking full axial views and applying a correction factor for magnification. In each of these cases a circular area of

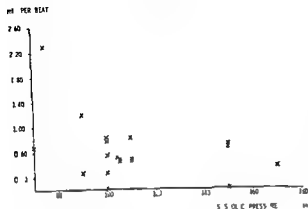


Fig 10 Volumetric displacement versus arterial systolic pressure in externa magna. At very low systolic pressure a sudden great increase in pulsation may be observed

8 to 9 mm diameter was subtracted from the cross-sectional area to represent the spinal cord and the validity of this diagram tested against the actual position of the spinal cord as shown on a film taken during encephalography at the time that the excursion of a fluid level was measured

Certain assumptions have been made in calculating the volume of CSF movement from the observed measurements (1) That the CSF alongside and in front of the cord is moving with the same amplitude of pulse as that revealed by the fluid level (2) That only a small portion of the systolic pressure rise in the CSF can be accommodated by compression of the air bubble (Boyle's law makes that evident)

Taking the cross-sectional area of the CSF space drawn on graph paper, and the amplitude of movement of the fluid level, it was possible to calculate the volumetric displacement of CSF up and down the spinal canal at this point with each heart beat

The mean of the observations 0.64 ml represents a very mixed group of patients under varying conditions of blood pressure. Statistical analysis is scarcely possible but if all the readings from all the patients are treated alike plotting of the figures for volumetric displacement against arterial diastolic blood pressure (Fig 9) arterial systolic pressure (Fig 10) and arterial pulse pressure (Figs 11 12) shows that the mean figure for CSF displacement is not dependent upon any of these but tends to remain constant (Fig 13)

This is confirmed by a detailed examination of the alterations in CSF displacement which occur in individual patients at the same examination. The volumetric displacement does not necessarily change at all with arterial blood pressure alterations nor when it does change, is the change always in the same direction

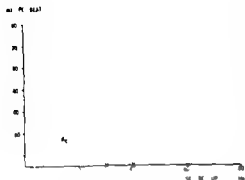


Fig 11

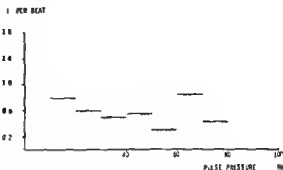


Fig 12

Fig 11 Volumetric displacement versus arterial diastolic pressure in cisterna magna. Pulse pressure and CSF displacement tend not to be related. The CSF volumetric displacement has a tendency to remain unchanged in single patients with varying blood pressure and from patient to patient under similar conditions.

Fig 12 Volumetric displacement versus arterial pulse pressure in cisterna magna. Mean values

This seems reasonable when it is considered that the CSF displacement at the cisterna magna represents the excess of cerebral arterial stroke volume over available intracranial elasticity. If auto-regulation is unimpaired and the pulse rate remains constant, the stroke volume should not be affected by blood pressure changes within certain limits.

The intracranial elasticity probably depends largely upon the compressibility of the venous vascular bed, and in any particular patient this will be dictated chiefly by the venous pressure in the head.

The available elasticity in the spinal canal also depends upon venous pressure but in the erect position is almost certainly always in excess of requirements. Consequently the CSF volumetric displacement at the cisterna magna in patients with auto-regulation intact is most likely to vary only with pulse rate and with the cranial venous pressure.

If auto-regulation is lost, however, there might be expected to be a variation with arterial blood pressure.

It is not, therefore, surprising to find that two patients in whom it was possible to measure a volumetric CSF displacement grossly in excess of the mean had extremely low arterial blood pressure. Under these circumstances the intracranial veins were probably collapsed. Moreover, auto-regulation may have been lost at this level of blood pressure.

On other occasions also a great increase in pulsation at the cisterna magna has been observed in conscious patients immediately before they have fainted. Measurement under such circumstances has, however, proved impossible.



Fig 13 The conus medullaris in an *Antilope cervicapra*. The theca outlined by contrast medium is a very narrow space. The spinal cord reaches the second sacral segment.

The volume of pulsatile CSF displacement here is therefore under the usual conditions during encephalography, about ten times greater than the displacement of fluid from the third (and probably the fourth) ventricle. Most of the upper cervical fluid pulse must come, therefore, not from the ventricular system but out of the basal cisterns.

Either by palpation of radial or carotid pulse and inspection of the movement on the monitor or by inspection alone to assess the type of movement it has been determined that in the great majority of sitting patients the CSF is thrust downwards during systole.

The usual caudal displacement of fluid seen in the sitting patient at encephalography as the brain expands, is also obvious when myodil droplets are carried to and fro in the prone position at myelography. The amplitude of

excursion in 8 normal cases ranged from 2 to 6.5 mm (mean 3.3 mm) in the canal at C3 and C4. It is estimated that the movements represent a mean volumetric displacement of at least 0.3 ml (range 0.2 to 0.8 ml). Myodil, however, is more viscous and has a higher specific gravity than CSF, so that the actual movement of CSF is probably greater than this calculation would suggest.

In the supine position, myodil may be made to outline the cerebellar tonsils, with the patient horizontal or head down. In the majority of normal cases, although the contrast medium may be seen to be washed backwards and forwards the profile of the tonsils themselves remains immobile. In only one case in whom no related abnormality could be found was any movement of the tonsils themselves seen. In this man a rhythmic downward movement was recorded coinciding in time with the systolic cranial pulse and having systolic thrust in a caudal direction.

As has been said the usual direction of movement of CSF is caudal in systole. On rare occasions however under as yet undefined conditions but always in horizontal or head down positions a reversal of the direction of movement at the foramen magnum may be observed. The myodil is thrust upwards into the foramen of Magendie, away from the spinal canal. The few cases with these unusual features are here described individually.

*Case 6* During myelography which proved to be normal myodil in the aqueduct of Sylvius was observed in the supine head down position. This myodil moved rostrally up the aqueduct during systole (timed by the carotid pulse). It also moved rostrally during inspiration and during abdominal compression.

*Case 7* The apparently normal patient mentioned above with the unusual tonsillar pulsation was also observed to fill his fourth ventricle with myodil from the cisterna magna when he coughed. (Tonsillar pulsation is seen in Chiari malformations but not usually in normal individuals.)

*Case 8* This patient with a normal myelography was suffering from severe headache while the following observation was made: contrast medium ran up the basal cisterns towards the cerebellopontine angle during systole while head down.

*Case 9* This patient had systolic pulsation up the aqueduct as judged by the type of movement and the radial pulse when supine horizontal and also 5° head down.

The inference of these observations must be that there are circumstances in which a cisternal driving force is sufficiently great to overcome the usually very considerably systolic brain expansion. In one case very complete observations were possible and it was seen that simultaneous with the spinal up-thrust into the fourth ventricle a caudally directed movement was taking place in the basal cisterns. In other words vascular expansion of the cerebrum seemed capable unexpectedly of forcing fluid into the fourth ventricle.

## Animal experiments

### *Cerebral arteries and CSF pressure*

*Arterial anatomy* Post mortem injections followed by radiography confirm the descriptions by TANDLER (1899) and HOFMANN (1900), among others, of the anatomy of extra and intra cranial arteries of a variety of mammals. Though the details of the arteries of the brain itself differ widely from species to species there is a strong resemblance of the general plan in all the species.

The extra dural supply to these arteries however differs in much more than detail. The interesting feature so far as CSF pulsations are concerned is the interruption of the arterial pathway to the brain by a rete which may alter the arterial pulse pressure and even the mean arterial pressure within the dura beyond the rete.

All mammals may be divided into three groups from this point of view (1) the Artiodactyla in whom the whole of the brain's blood supply passes through retia, (2) the Felidae in whom a great proportion of the cerebral blood has to travel via retia, and (3) nearly all the rest in whom there is no such interruption.

Mammals also differ one from another in brain size and thus quantitative arterial inflow to the head with each heart beat.

Thus one might expect to see variations in both pulsation amplitude and mean pressure of CSF between the three groups, and brain size will also affect the amplitude of CSF movement. Both Artiodactyla and Felidae have a system different from man so that something may be learned from the comparison.

*Group 1 (Artiodactyla)* In the goat (*Capra hircus*), CSF pressure (measured under general anaesthesia with halothane 1.5 % and oxygen via an endotracheal tube) by cisternal puncture with the animal lying on its side, varied between 30 and 80 mm H<sub>2</sub>O (mean) and showed a respiratory variation of at most 40 mm H<sub>2</sub>O but a cardiac variation of less than 20 mm H<sub>2</sub>O.

*Group 2 (Felidae)* In the domestic cat (*Felis catus*), cerebral blood supply is only partially supplied via a rete. Animals anaesthetized with leep doses of pentothal only had a CSF pressure up to 120 mm H<sub>2</sub>O peak measured, as in the goat recumbent, at the cisterna magna. Cardiac variation lay between 20 and 60 mm H<sub>2</sub>O and respiratory variation was 180 mm H<sub>2</sub>O.

*Group 3* Other carnivora without retia which have been examined (sun bear — *Helarctas malayanus* for instance) exhibit CSF pressure of the same order as man 20/0 cm H<sub>2</sub>O (10 cm H<sub>2</sub>O cardiac variation, 10 cm H<sub>2</sub>O respiratory variation). Anaesthesia given by an endotracheal tube was halothane 2.0 % and oxygen.

Table

*Measurements of abdominal and thoracic vena caval pressure*

Species	Abdominal posterior vena caval pressure (mm H <sub>2</sub> O)	Thoracic posterior vena caval pressure (mm H <sub>2</sub> O)
<i>Oryz. lagus cuniculus</i>	60-30 (measured posterior to renal area)	70/-37
<i>Felis catus</i>	40-19 (post renal)	44/-22
<i>Canis latrans</i>	0- (hepatic region)	15/-2
<i>Canis lupus</i>	Atmospheric (post renal)	0/-50
<i>Capra hircus</i>	Atmospheric (no variation up to renal)	34/-10
<i>Erythrocebus patas</i>	40-80	0/-120

The Patas monkey also exhibits CSF pressures measured in the lateral ventricles and at the cisterna magna which are very similar to those in man.

*The posterior vena cava* The importance of epidural venous pressure has often been emphasized in the literature concerned with CSF pressure. Indeed the CSF pressure and the venous pressure must always be almost identical. Differences between them described or implied in this investigation can only be of a few millimetres of water.

Spinal epidural veins in the lumbar region are connected by their tributaries to the posterior vena cava. The vena caval pressure must therefore presumably affect most directly the degree of turgor in these epidural veins and secondarily through them the CSF pressure (except perhaps in some diving mammals in which the spinal veins may be widely separated from the theca).

It appears from the work of FIENNES DU BOULAY, GABRIEL & VERITY (unpublished) that many pronograde mammals appear to have mechanisms at the level of the diaphragm which alter and diminish the transmission of intra-thoracic pressure swings backwards along the vena cava but these mechanisms are effective to different degrees (see Table).

In *Capra hircus* it should be noted that in the vena cava posterior to the entry of the renal veins the pressure was zero and almost non-pulsatile.

In *Felis catus* on the other hand, the vena caval pressure and pulse pressure was considerable and there was no mean pressure drop along the abdominal vena cava.

It should be noted that under the particular conditions of the experiments (animal supine and anaesthetized) the CSF pressure generally fell on inspira-

tion as did the posterior vena caval pressure. Abdominal compression caused both to rise (except in the sea lion).

In the *Erythrocebus patas* the posterior vena caval pressure swing closely reflected the intra thoracic pressure swing, in which it resembles man. There is a difference only in mean pressure between abdomen and thorax.

The inter relationship between intra cranial arterial pressure, epidural venous pressure and CSF pressure and movement in *Felis catus* is still obscure, but in *Capra hircus* the fact that the CSF is non pulsatile fits simply with the observed vascular anatomy and pressures.

### *CSF pulsatile movements*

In six rabbits myodil was introduced into the cisterna magna or the lateral ventricles. No pulsatile movements were seen under anaesthesia in the cisterna magna, cervical spinal canal, ventricles or basal cisterns. The rabbit's brain is however comparatively small and movements if they occur, would be expected to be of small amplitude.

In *Felis catus* pulsatile movements of myodil in the cervical spinal canal are barely detectable.

In the *Canidae* examined pulsatile movements were seen without difficulty.

In *Capra hircus* no pulsatile movements were detected under any circumstances neither with the animals anaesthetised nor awake, lying nor standing, neither in the spinal canal nor the basal cisterns.

In the normal *Erythrocebus patas* pulsatile movements occur very much as in man. Myodil introduced into the lateral ventricles is almost immobile but once it has passed through the foramen of Monro, to and fro movements are always seen round the large massa intermedia and down the aqueduct of Sylvius. Movements may also be seen in the fourth ventricle and in the basal cisterns. The thrust is usually systolic in timing down the aqueduct and down the basal cisterns towards the foramen magnum. In the cervical spinal canal it also has a caudal systolic thrust.

In one *Patas* monkey as in a few adult humans in the horizontal or head down position a systolic thrust was seen up the aqueduct for a period of about 10 minutes while the myodil passed from third to fourth ventricles under the influence of the flow of CSF, impeded as it was by the regular pressure wave passing upwards against the slow stream. Abdominal compression caused the myodil to pass back up the aqueduct for a few millimetres.

At the foramen magnum itself the anatomic relationships cannot exactly correspond to the human situation, however since the cerebellum does not entirely cover the medulla and the cerebellar tonsils do not reach quite so far caudally as they do in man.



*Discussion of animal experiments*

It may be assumed as in man that rapid movements in the CSI are chiefly the result of arterial pressure in the head but modified greatly by the nature of the total cerebral blood flow by the compressibility of the cranial venous bed and the spinal epidural venous plexus and modified also by variations in the size of CSI pathways.

Whereas the anatomy of the circle of Willis and its branches is remarkably similar in all the mammals investigated there are basic differences proximal to the circle which must modify the pulsatile nature of the cerebral blood flow. WILKES (1691) probably understood correctly the effect of the rete in smoothing the pulsatile nature of the flow and in some instances lowering its pressure.

Both theoretical and experimental observation lead to the conclusion that in Artiodactyla and perhaps in Felidae the rete caroticum diminutivum has the pressure of the cerebrospinal fluid.

Skull films of about 200 species of mammal have been made. Not only do many non primates have bony tentoria but the convolutional impressions caused by the cerebrum are often strikingly obvious over the vault as well as the skull base even in the adult as state of affairs which differs from man. In humans the upper part of the vault tends to be separated from the cerebrum by a layer of CSI diminishing the influence of the shape of the brain upon the inner table of the skull in this region (DU BOUISSAY 1956). The necessity for this space filled with CSI is probably related to the large size of man's brain and the considerable alterations in brain volume which must accompany large changes in cerebral blood flow. It is tempting to look at the disadvantages of a deep CSI layer — the likelihood of displacement and torsion injuries, perhaps added risk of subdural haemorrhage. The probable role of the rete in diminishing brain pulsation would remove in those animals that possess a rete at least the requirement for the cranial cavity to be large enough to accommodate pulsatile brain expansions.

Pulsatile pressures require elasticity to be present before movement can take place and much of this elasticity has been shown in man and dogs to reside in the spinal epidural veins. Were no pulsations present a turbid system of spinal veins might be less essential and in proneigrade animals this too might be an advantage since the heart in them is lower than the spine when they stand making the maintenance of a positive venous pressure in the lumbar region problematical.

In spite of all these considerations the advantage in abolishing the rather small pulsations seems perhaps not great enough to explain the evolutionary trend towards retria which is evident in the Eocene period when Artiodactyla

and Felidae seem to have appeared and the, perhaps more recent, adaptation of Prosimians to a similar mechanism.

Consideration of the CSF space in man reveals that not only the cortical spaces but also the lumbar and lumbo-sacral theca are extraordinarily capacious. A possible reason for this may be that great movements of CSF take place from head to spinal theca and back due to alterations in total cerebral blood flow. As is well known a rise in arterial  $p_{aCO_2}$  tension causes swelling of the brain. A fall shrinks the brain. The changes are probably considerable even within the physiologic range and appear to be due basically to an alteration in the calibre of all the small blood vessels which dilate to increase the cerebral blood flow as the  $p_{aCO_2}$  rises. In man the brain is so large that even quite small alterations in regional circulation if they take place in many regions simultaneously, must substantially affect the total volume of the brain's blood supply.

The changes which must take place during sleep and the pathologic but recoverable changes accompanying for instance respiratory disease, will be accommodated by the displacement of CSF from the one large reservoir to the other.

It seems possible that the presence of considerable cardiac and respiratory pulsations in the CSF of primates and some other large mammals are a side effect of the necessary size of the CSF reservoir.

The size of the spinal reservoir has been investigated radiographically in a selection of mammals by introduction of contrast medium and while no volumetric figures are available the visual impression provides striking confirmation of the radical difference between Artiodactyla and animals without a rete cerebri. In all the Artiodactyla examined the spinal cord comes right down to the lower end of the spinal canal where it stops abruptly (Fig. 13). There is no room for any large spinal CSF reservoir. In those with arterial retia interposed between the neck arteries and the intracranial vessels it may be that the function of the rete is not merely to lower the intracranial blood pressure and pulse pressure but perhaps more important to take away from the intrinsic vessels of the brain a major share in the control of total blood flow. In that way substantial alterations in total cerebral blood flow should be possible without substantial alteration of volume of the brain by its arterioles.

### Conclusions

Certain mammals may have evolved a system of cerebral blood flow which is dependent upon and mutually linked with adaptations in the CSF hydrodynamics and abdominal venous pressure.

## ZUSAMMENFASSUNG

Messungen über die Bewegungen von Luft und positivem Kontrastmittel während der Encephalographie, Ventriculographie und Myelographie wurden bei 190 Patienten und einer Anzahl von Kaninchen, Katzen, Ziegen und Affen vorgenommen. Druck und Stromung in der Vena cava posterior wurden in ähnlichen Tieren untersucht und die Anatomie der Gehirnarterien an 110 Tieren studiert. Die Ursachen und Amplituden der pulsformigen Bewegungen der Zerebrospinalflüssigkeit wurden beschrieben und deren Bedeutung vom Gesichtspunkt der zerebralen Durchblutungskontrolle spekulativ betrachtet.

## RÉSUMÉ

Les auteurs ont mesuré sur 190 malades et sur un certain nombre de lapins, de chats, de chèvres et de singes les mouvements de l'air et des moyens de contraste positifs au cours de l'encéphalographie, de la ventriculographie et de la myélographie. Ils ont aussi étudié sur des animaux semblables la pression et le débit dans la veine cave postérieure et l'anatomie des artères crâniennes sur 110 espèces de mammifères. Ils décrivent les causes et l'amplitude des mouvements pulsatiles du liquide céphalo rachidien et font des hypothèses sur leur influence du point de vue de la régulation du débit sanguin cérébral.

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## HYDROCÉPHALIE POST TRAUMATIQUE ET MESURE DE LA PRESSION DU LIQUIDE CÉPHALO RACHIDIEN

par

G. IORIOU et F. ZANDER

Comme nous l'avons énoncé lors d'un précédent travail (ZANDER & IORIOU 1963) l'hydrocéphalie est de règle chez tout traumatisé crânio-cérébral grave resté plus d'une semaine dans le coma.

Cette hydrocéphalie est généralisée et commune avec une progression est rapide dans certains cas, et en rapport direct avec l'âge du malade. Elle atteint, en général son maximum de développement dans les cas restés longtemps dans le coma mais elle peut parfois doubler de volume entre la 1<sup>ère</sup> et la 2<sup>ème</sup> semaine post traumatique (Fig. 1).

Nous avons également remarqué que l'IICG utile pour le pronostic vital de ces cas ne apporte pas de renseignement concernant l'évolution de l'hydrocéphalie. En ce qui concerne les lésions pathologiques elles sont d'ordre myélinolytique.

Nos expériences concernant le problème thérapeutique de l'hydrocéphalie post traumatique les différentes indications opératoires ainsi que les modifications échographiques et l'apport de la cisternographie aux isotopes ont fait l'objet des travaux antérieurs (ZANDER et coll. 1968, IORIOU 1970). Par la même occasion, nous avons esquissé une hypothèse concernant la pathogénie de l'hydrocéphalie post traumatique.

Le but de ce travail est de rapporter les résultats des mensurations de la pression du LCR, dans les cas d'hydrocéphalie post traumatique et en discuter



Fig. 1 Installation et progression d'une hydrocephalie interne post traumatique. Quatre sept dix huit et vingt trois semaines apres traumatisme cranio-cerebral

nos observations. Ces mensurations ont été effectuées à trois endroits différents: le ventricule latéral, l'espace epidural cérébral et la région lombaire.

**Méthode.** Pour mesurer la pression du LCR intra ventriculaire, nous avons utilisé d'abord la méthode manométrique et par la suite électronique. Un drain ventriculaire connecté à un réservoir de Rickham (identique à celui employé pour le drainage ventriculo-auriculaire des cas d'hydrocephalie) est placé soit dans la région frontale soit dans la région pariétale. La ponction du réservoir de Rickham est faite perpendiculairement au capuchon du réservoir et à l'aide d'une aiguille No 19 ou 20. La lecture de la pression de la chambre du réservoir donne indirectement la valeur de la pression intra ventriculaire.

Le même dispositif a été aussi utilisé pour la lecture de la pression epidurale de la région pariétale. Dans ce cas précis on prend soin d'injecter dans le système drain-réservoir une petite quantité d'eau (puisque normalement il n'existe pas de liquide dans cet espace). L'amplitude de la compression du drain par les pulsations cérébrales donne indirectement la valeur de la pression qui règne à cet endroit.

Quant à la lecture de la pression lombaire elle se fait directement en connectant l'appareil enregistreur à l'aiguille de la ponction lombaire (pour plus de détails nous prions le lecteur de se référer à notre travail, FOROGLIOU 1970).

## Resultats

### *Patient à l'état de veille et au repos*

**Pression au niveau des ventricules.** La pression a été enregistrée sous forme d'ondes pointues et en dents de scie. Les ondes sont dichrotiques dans les deux sens périodiques, d'une grande régularité et en rapport étroit avec le pouls artériel. La pression est en général stable mais elle montre le plus souvent des excursions dans les deux sens (augmentation et diminution).

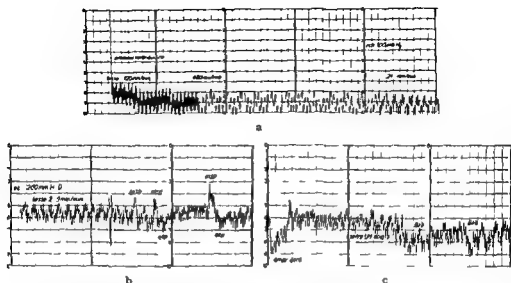


Fig. 2. Représentation graphique de la pression intra ventriculaire chez un patient hydrocephale traité par dérivation ventriculo-audiculaire (méthode de Spitz Nulsen avec valve de Holter) a) Patient éveillé et au repos b) Influence de la respiration forcée sur la pression intra ventriculaire c) Influence des différentes manœuvres sur la pression intra ventriculaire

Lors de l'enregistrement à la petite vitesse on peut encore démontrer un mouvement ondulatoire de la courbe enregistrée, qui est probablement en relation avec les mouvements inspiratoires. Un analyseur de fréquence nous donnerait davantage de renseignements mais nous n'en disposons pas encore (Fig. 2 a)

**Pression au niveau de l'espace épidural** L'enregistrement dans cet espace recueille des ondes qui ont le même aspect que celles décrites dans le système ventriculaire quoique moins amples.

Les valeurs obtenues sont identiques à celles captées à l'intérieur du système ventriculaire.

**Pression au niveau lombaire** La courbe obtenue n'est pas linéaire elle est par contre constituée par une succession des points sous forme d'escalier avec un rythme correspondant à celui de la courbe obtenue dans le système ventriculaire, la fréquence étant synchrone au pouls artériel.

#### *Influence des différentes manœuvres sur la pression*

**La respiration** Comme LUNDBERG (1960) l'a déjà signalé, il existe une relation directe entre la fréquence respiratoire et le changement rapide de pression du liquide qui caractérise les ondes A décrites par lui.

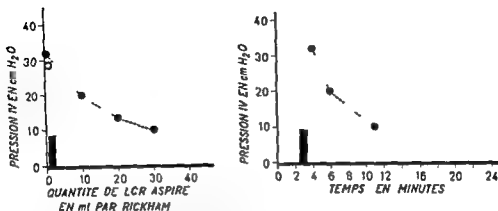


Fig 3 Représentation graphique de la pression intra ventriculaire avant et après traitement de l'hydrocéphalie interne. O pression lombaire ■ pression ventriculaire 12 mois après intervention constante lors de contrôles ultérieurs

Cette relation n'est d'ailleurs pas une condition nécessaire pour l'apparition de telles ondes. Pour cet auteur, l'hyperpnée peut provoquer une chute rapide de la pression intra ventriculaire, alors que l'hypopnée provoque une augmentation de cette pression par un mécanisme de relation respiratoire tension CO<sub>2</sub> artériel.

De même, nous avons remarqué lors de l'inspiration profonde une chute de la pression intra ventriculaire d'une à deux unités et en même temps une diminution de la fréquence des ondes enregistrées. L'expiration provoque l'effet contraire, elle est suivie immédiatement d'une normalisation de la fréquence des ondes inscrites (Fig 2 b).

**La pression de l'artère carotide.** La courbe enregistrée montre un pic immédiat de la pression intra crânienne suivie d'un ralentissement brusque du rythme enregistré. À la fin de la compression, il y a une chute brutale de la pression intra-ventriculaire, probablement compensatrice avec normalisation rapide de la courbe enregistrée (Fig 2 c).

**Différentes manœuvres neurologiques et neuro psychologiques.** Elles ne modifient que peu la pression intra ventriculaire. En effet, lorsqu'on demande au patient de serrer les doigts de l'examineur (épreuve de force), il n'y a pas de changement de la pression intra ventriculaire, il existe peut-être une discrète modification du rythme enregistré (Fig 2 c).

La recherche du signe de Babinski modifie l'amplitude des ondes avec des valeurs extrêmes. Mais il faut se rappeler que la recherche d'un tel signe peut provoquer dans certains cas des mouvements brusques de retrait de la jambe, ou



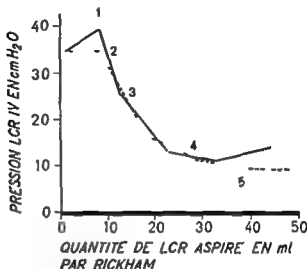


Fig 4 Étude simultanée des pressions intra ventriculaires et lombaires dans un cas d'hydrocéphalie post traumatique — Rickham lombaire

même un sursaut de tout le corps ce qui peut modifier la pression intra ventriculaire et provoquer même des artefacts (Fig 2 c)

Enfin des calculs simples effectués par le patient par cœur (pour éviter des mouvements lors de l'écriture et ainsi des artefacts) ne modifient guère la courbe enregistrée

### Discussion

Comme nous l'avons noté précédemment (ZANDER & FOROGLU 1969), il existe dans les cas post traumatiques frais une élévation de la pression du LCR au niveau lombaire. Une semaine après le traumatisme, la pression lombaire devient normale, pour augmenter à nouveau dans les cas qui passent à la chronicité (persistance du coma). On finit ainsi par avoir dans les cas chroniques une hydrocéphalie interne post traumatique communicante associée à une hypertension du LCR et ceci en absence d'autres signes cliniques d'hypertension intracranienne.

Il est aussi connu qu'en cas d'hydrocéphalie interne importante les espaces sous-arachnoïdiens de la convexité du cerveau ne sont pas visualisés lors de l'encephalographie. En revanche il y a parfois de l'air dans les citernes de la base. L'air ou le gaz injectés se localisent surtout dans le système ventriculaire qui apparaît plus ou moins dilaté suivant les cas.

Nous avons voulu examiner de plus près la raison pour laquelle le produit injecté était en quelque sorte aspiré par le système ventriculaire dilaté.

La cause de la seule insufflation du système ventriculaire pourrait être une pression différentielle importante entre le système ventriculaire et le système sous-arachnoidien, soit par bloc organique de ce dernier (adhérences), soit par trouble fonctionnel du liquide circulant dans les espaces sous-arachnoidiens ce qui ne permettrait pas leur visulisation lors de l'encephalographie gazeuse.

C'est la raison de la mensuration de la pression du LCR dans les différents compartiments.

Nous avons ainsi trouvé que dans les cas étudiés il existe une pression intra-ventriculaire élevée et ceci en absence des signes cliniques d'hypertension intracranienne. Dans les cas d'hydrocephalie non traitée les valeurs varient entre 20 et 40 cm d'eau alors que dans les cas qui ont bénéficié d'un traitement chirurgical la pression est inférieure à 10 cm d'eau (Fig. 3).

La mensuration simultanée du liquide de la région lombaire a donné des valeurs légèrement inférieures à celles obtenues au niveau ventriculaire (Fig. 4).

Quant à la pression épidurale cephalique les valeurs obtenues après traitement de l'hydrocephalie ont de la même grandeur que celles du système ventriculaire.

L'interprétation des courbes obtenues est difficile elles ne ressemblent pas à celles décrites dans la littérature. En effet celles enregistrées par LUNDBERG (1960) et LUNDBERG et coll (1965) ont en plateau.

LAITINEN et coll (1966) et LAITINEN (1968) ont enregistré lors des opérations stéréotaxiques certaines courbes du liquide céphalo-rachidien elles ont également en plateau mais elles montrent aussi un certain dichotisme.

En revanche nos courbes montrent une périodicité synchrone au pouls radial et un dichotisme.

Quant à l'origine des pulsations enregistrées les idées des différents auteurs sont partagées. Si l'origine veineuse n'a plus beaucoup d'adeptes actuellement les discussions persistent sur l'origine artérielle à savoir si c'est le plexus choroïde seul qui est responsable des pulsations du liquide ou toutes les artères de la base du cerveau en étaient la cause (O'CONNEL 1943 citée par LAITINEN 1968). D'après ce dernier auteur l'origine des pulsations était extrachoroïdienne et certainement artérielle.

## RÉSUMÉ

Les patients victimes d'un traumatisme crânio-cérébral grave restent longtemps dans le coma développent une hydrocephalie interne communicante généralisée et progressive. La pression du liquide céphalo-rachidien est augmentée aussi bien dans les cas chroniques que dans les cas aigus. Cette pression est augmentée dans tous les compartiments examinés : système ventriculaire, espace épidural pariétal et région lombaire. Différentes courbes ont été enregistrées dans les régions précitées à l'état de veille et lors des différentes manœuvres.

## SUMMARY

Patients who have sustained severe brain injury and suffer prolonged coma usually develop internal communicating hydrocephalus of a general and progressive nature. The intracranial pressure is raised both in the acute and chronic stages and may be measured in the ventricles, the epidural space and the lumbar region. Pressure curves were obtained in all these areas under a variety of conditions while the patient was conscious.

## ZUSAMMENFASSUNG

Patienten mit einer schweren Hirnverletzung und die lange bewusstlos waren, entwickeln einen internen kommunizierenden Hydrozephalus, der generell und progressiv ist. Der Hirndruck ist gesteigert im frischen und im älteren Fall. Diese Drucksteigerung umfasst die Ventrikel, den parietalen Epiduralraum und die Lendenregion. Druckkurven in den erwähnten Orten wurden während des Wachzustandes unter verschiedenen Bedingungen registriert.

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## RISA CISTERNOGRAPHY IN POSSIBLE HYDROCEPHALUS

### Diagnostic and therapeutic experiences

by

H FORSLO B FORSSMAN S JARPE and C RADBERG

Possible alteration in the cerebrospinal fluid circulation mainly in dementia, idiopathic or secondary to a definite etiology, e.g. subarachnoid hemorrhage, trauma of the head or meningitis have suggested isotope cisternography in conjunction with more traditional neuroradiologic procedures (Di Chiro 1964). The purpose of this paper is to correlate the cisternographies especially those indicating ventricular activity with the encephalographies and angiographies. A Spitz Holter ventriculo-atrial shunting procedure was considered desirable in 22 of the 51 patients of this material. Prognostic criteria for the operation are formulated from the experiences gained.

*Method* All cisternographies in adult patients of this series were carried out by injecting 100  $\mu$ Ci RISA in a volume of 7 ml later reduced to 3 ml into the lumbar subarachnoid space. The total protein content never exceeded 2 mg. The puncture was performed with a small spinal needle (20 to 22 gauge) to minimize leakage of activity from the subarachnoid space. The first patients of the series were injected sitting and in some the subarachnoid position of the needle was confirmed by injection of a few ml of air. The incidence of leakage with this technique was, however, fairly high, possibly due to the increased

hydrostatic pressure the method was therefore changed to an injection in the prone position during TV screening so as to secure correct positioning of the needle tip. After injection the needle was kept in position for 5 minutes to reduce leakage through the puncture hole and give any raised injection pressure time to subside. The patients were kept prone until the two hour documentation began. This technique has virtually eliminated the incidence of leakage.

Registrations were performed in the upright and left lateral positions at 2, 6 and 24 hours after injection with additional scans at 30, 48 and 72 hours or even later when the flow was slow. Vertex projections were often included and registration of the activity in the lumbar region was made almost always at the beginning of the investigation to demonstrate possible activity outside the subarachnoid space. A 5' Magna scanner or a Nuclear Chicago Pho-Gamma VI was used for scintigraphy. Encephalography ad modum LINDGREN (1949) was performed. Specific attempts were made to facilitate gas filling of the extra-ventricular cerebrospinal fluid pathways. The hydrocephalic index as proposed by LINDGREN (1951) was calculated and the temporal horn indices were determined by the method proposed by SJAASTAD et coll. (1969). Cerebral angiography was carried out by percutaneous puncture of the common carotid artery and the introduction of a catheter into the common or the internal carotid artery. Atrio-ventricular shunting when subsequently performed consisted of implanting a Spitz-Holter medium pressure shunt valve with its attendant tubing by the technique described by two of the authors (JARPE et coll. 1969). This type of valve was arbitrarily chosen. Care was taken to position the tip of the ventricular catheter in the right frontal horn, thus avoiding blockage in the catheter by the choroid plexus.

Shunt failure appeared clinically possible in 2 patients but reoperations proved both shunts to be functioning correctly. The valve in one of the patients was however changed to a Holter low pressure type although this had no effect on the patient's progressive dementia.

*Material.* A total of 66 patients underwent cisternography during the period 1968–1970 after eliminating technically unsuccessful and less relevant cisternographies. 51 patients, 36 males and 15 females, remained. The sex and age distribution is given in Table 1 and other data concerning the patients and investigations in Table 2.

*Neurologic symptoms and signs.* The signs of pre-senile or senile dementia with their counterparts of normal pressure hydrocephalus or cerebral atrophy in this material present fairly uniform agreement with previous reports. However, as far as symptoms are concerned the order of first appearance, the number, the severity and the speed of progression may vary. The main features are mental

Table 1

*Sex and age distribution*

Age ranges	Males	Females	Total
60—75	13	5	18
30—59	19	7	26
10—29	3	2	5
< 10	1	1	2
Total	36	15	51

defects usually starting as poor recent memory and followed by reduction of activity obvious dementia and disorientation with absent or limited comprehension. Disturbances of gait similar to the customary ataxia of the aged or corresponding to the term gait apraxia appear; this term means difficulty in initiating and stopping walking movements (long tract signs), almost synonymous to spastic paresis usually bilateral and with predominance of spasticity become evident. Finally urinary with sometimes rectal incontinence, may develop. The differentiation of normal pressure hydrocephalus from cerebral atrophy may not be possible from the clinical signs.

A normal pressure hydrocephalus may progress so slowly over years as to make differentiation from cerebral atrophy hazardous in cases of unknown etiology. Arnold Chiari malformation and an elongated basilar artery. In cases with known acute incidents such as subarachnoid hemorrhage, severe head trauma and meningitis the progression of normal pressure hydrocephalus is usually more rapid with definite signs developing in the course of some weeks or months.

*Results from surgical intervention.* The relatively small material and lack of equipment for digital analysis of the Gamma camera images preclude any attempt being made to categorize ventricular isotope filling into various groups or patterns. As cisternography was used as an aid to select patients who would benefit from ventricular shunting the main interest was focussed on whether ventricular activity appeared or not; a shunt was not deliberately attempted if no ventricular activity was observed. There are two exceptions to this (cisternographies 1 and 3). At the first cisternography, which was unsatisfactory it was wrongly concluded that ventricular activity had appeared. The other case was shunted to relieve intermittent obstruction by a large cavum septi pellucidum. Fifteen patients of the present series were operated upon with implantation of a ventriculo-atrial Spitz-Holter shunt subsequent to cisternography. Another

Table 2

*Survey of material—Code list*

1 Consecutive number of cisternographies	13 Hypertension
2 Age	14 Arteriosclerosis of cerebral vessels at angiography
3 Sex	15 Chronic alcoholism
4 Arnold Chiari malformation	16 Dementia
5 Juvenile hydrocephalus	17 Ataxia
6 Tumour or cyst	18 Long tract signs
7 Infectious arachnoiditis	19 Epilepsy
8 Subarachnoidal hemorrhage (aneurysm)	20 Circulation time carotid angiography
9 Subarachnoidal hemorrhage unknown source	21 Index anterior horns (two values when marked difference in width)
10 Severe head trauma	22 Width of third ventricle temporal horn index (TV/TH)
11 Elongated basilar artery	
12 Cerebral atrophy histologically verified	

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1	54	f									x				x				1
2	12	m							x						x			x	1
3	23	f				x	x												4
4	41	m													x		x		5 1/2
5	38	m							x										3 1/2
6	73	m								x		x	x			x			
7	13	f						x							x	x			4 1/2
8	52	m											x		x	x			5 1/2
9	60	m							x	x		x	x	x	x	x			7
10	44	m						x							x	x			4 1/2
11	31	f																x	4
12	42	m							x										4
13	52	m							x						x	x			4 1/2
14	22	m					x								x		x		5 1/2

Table 2 (cont.)

- 23 Width of right and left temporal horn index against lateral ventricle (LV/TH)
- 24 Degree of extracerebral gas filling during cisternography  
*Convexity gas*  
 0 complete block over convexity  
 1 gas filling of single furrows usually at interhemispheric fissure  
 2 gas filling of Sylvian fissure but not parasagittally  
 3 normal gas filling of extracerebral fluid pathways
- 25 Isotope activity in ventricular system 0 or <
- 26 Shunting procedure performed shunt later removed x0
- 27 Clinical course according to HAALAND & LOVSTAD (1970) as follows  
 0 no dementia working full time  
 I working part time  
 II unable to work manages daily routine without help  
 III needing help with daily home routine in a rest home or equiv  
 IV needing full hospital care
- 28 IQ (intellectual quotient)
- 29 Shunted before cisternography

21	22	23	24	25	26	27	28	29
		6 40						
0 37	12 9 2	5 22	3	0	<	III—III		
		8 30						
0 38	13 19	6 35	2	0	0	I—I	70	
		3 50						
0 34	12 30	5 36	3	0	x	0—0	12b	
		4 45						
0 38	14 31	5 42	3	II	0	II—II	73	
		4 28						
0 34	9 15	8 18	3	0	0	C—C		
		4 55						
0 34	18 36	6 43	2	x	x	II—III		
		II 28						
0 29	12 15	8 24	0	<	<	IV—0	65	
		5 54						
0 40	14 28	5 52	2	x	x	III—IV		
		10 25						
0 42	III 17	11 22	2	x	<	III—II	88	x
		12 18						
0 42	15 10	17 16	0	x	<	III—0	10a	
		3 63						
0 31	15 38	5 44	2	<	x	0—0		
		5 32						
0 31	9 16	6 28	0	0	0	0—0		
		16 31						
0 60	III 11	16 31	2	x	x	II—IV		
		13 19						
0 38	17 12	16 13	1	x	x	IV—IV		



Table 2 (cont.)

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
15	13	m		x	v													x	
16	45	f					v								v				5
17	33	m																	3 1/2
18	61	m							v						v	v			
19	40	f	v														v		3 1/2
20	45	m						v				v				x			3 1/2
21	71	m									x				x		v		7
22	58	m							v						v				6
23	50	f										x			v				7
24	51	m			v						x								6
25	55	m							x									x	
26	56	f													v		v		
27	56	m										x	x		v	x	v		4 1/2
28	61	m	x													v		x	
29	3	m																	
30	60	f																x	
31	41	m																	
32	5	f																	
33	56	m								v		v	v						
34	66	m										v	v		x	v		v	6
35	65	f							v		x				x	v			
36	54	m										x			x	x		v	7 1/2
37	56	m								x		x	v		x	x			6
38	41	m		x															5 1/2
39	75	m												v					10

Table 2 (cont.)

21	22	23	24	25	26	27	28	29
085		12 46						
045	25 23	10 42	0	x	x	0-0		x
		13 22						
046	12 11	8 29	0	x	x	IV-IV		
076		4 32						
037	9 26	3 67	3	0	0	II-II		
		4 62						
034	16 20	12 25	2	x	x	II-II		
		6 20						
029	10 17	6 22		0	0	0-0		
				0	0	II-II		
		14 21						
046	16 09	20 19	2	x	x	IV-III		
				x	0	IV-IV		
				0	0	IV-III		
		11 26						
040	16 11	19 19	1	x	x0	III-III		x
		2 105						
033	12 4	4 60	1	x	0	I-I	114	
		6 28						
035	15 16	13 15	2	0	0	I-II	died	
		3 80						
037	13 37	4 60	2	0	0	II-II		
		8 34						
044	14 19	7 39	3	0	x	III-III		x
		2 85						
032	11 55	2 80	3	0		0-0		
		6 37						
039	15 27	5 42	2	0	0	0-0	112	
		2 55						
076	11 20	4 28	3	0	0	0-II		
		6 37						
040	15 21	11 30	3	0	0	0-0		
				0	0	II-II		
		5 40						
035	13 24	6 40	2	x	x	III-III		
		4 67						
040	18 33	7 37	2	x	x	III-II		
		11 27						
043	18 16	11 29	2	x	0	III-III	112/99	x
		9 28						
047	16 20	7 34	2	x	x	III-III		
047	20 15	13 25	1	x	x	0-II		x
				x	0	III-III		x

Table 2 (cont)

I	II	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
40	74	m										x						x	4
41	62	f													x				
42	61	m						x											5
43	52	m						x						x	x				
44	64	m									x							x	4
45	39	m			x														3 1/2
46	18	f	x														x	x	
47	64	f													x		x		5
48	12	m																x	
49	63	m										x	x	x			x		
50	65	m										x							
51	55	f								x		x	x		x	x	x		5 1/2
52	41	m													x	x	x		

eight cisternographies were performed in patients previously so operated (One patient figures in both groups: cisternographies 13 and 43). Twelve patients (9 from the former group, 3 from the latter) out of this total of 22 patients form a distinct, clinically homogeneous group. They all presented with rapid progressive dementia and ventricular enlargement; mechanical obstruction other than cisternal blockage (e.g. tumours, cysts, Arnold-Chiari malformations) was proved to be absent. The pre- and postoperative courses of this group related to etiologic factors behind the ventricular enlargement are presented graphically in Fig. 1.

It is obvious that all patients with subarachnoidal bleeding have responded well after shunting, 2 of them returning to full time work. The patient with cisternography 9 — a chronic alcoholic — has returned to his pre-morbid level. The patient with cisternography 16 is on her way to recovery, delayed, however, by a left internal capsule lesion with hemiparesis and aphasia. The remaining 10 patients form a very heterogeneous group and do not readily lend themselves to collective analysis; only 2 of them have undergone permanent postoperative

Table 2 (cont.)

21	22	23	24	25	26	27	28	29
		4 6 0						
0 41	18 3 6	III 4 3	1	0	0	0-0	98	
		6 3 7						
0 36	14 2 8	4 5 2	3	0	0	III-III		
				x	x	IV-IV		
		16 3 1						
0 60	18 1 1	16 3 1	2	v	v	IV-IV	14/69	x
				0	0	II-II		
		11 3 1						
0 50	25 2 0	12 3 1	0	x	0	II-II	III	
			3	0	0	0-0		
0 41		8 3 1						
0 36	15 2 3	5 3 6	2	v	0	IV-IV		
		3 3 7						
0 32	8 2 3	4 3 5	3	0	0	0-0		
		9 2 4						
0 41	14 1 4	11 2 0	1	x	0	II-II		
		8 3 1						
0 41	16 1 9	9 9 6	1	x	0	0-0	104	
		5 9 2						
0 45	24 4 4	6 7 0	2	v	v	III-III	64	
		7 3 4						
0 36	18 2 8	6 3 7	3	v	0	II-II	77	

improvement and to a small degree one moving from group III to II, the other from IV to III. The remaining patients in this group are unchanged or worse. Details concerning their histories, clinical findings and postoperative courses may be extracted from Table 2 (patients with cisternographies 3, 6, 11, 14, 15, 24, 28, 34, 38 and 42).

The present material of patients subjected to cisternography form but a small portion of the total number of demented, hydrocephalic adults shunted at the neurosurgical clinic. Experiences of this larger group produce the same conclusion as indicated above: shunt operations will be successful in demented, hydrocephalic adults with a verified history of arachnoiditis (JARPE 1970).

*Correlation between neuroradiologic findings and operative result.* It is well known to all investigators who have used isotope cisternography that far from all patients with ventricular activity will respond with improvement upon ventricular shunting. On comparison with other neuroradiologic examinations it was evident that improvement occurred exclusively in patients who at cisternography had a certain dilatation of the temporal horns as evidenced by the

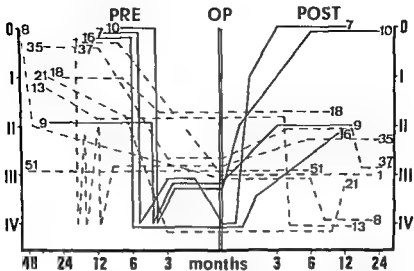


Fig 1 Pre and postoperative courses in 17 Spitz-Holter shunted patients with presenile dementia grouped according to the etiologic factors in their histories: — Subarachnoidal bleeding verified by lumbar puncture — Cerebral atrophy according to brain biopsy at shunt operation — Obsolete etiology

two kinds of temporal horn index proposed by SJAASTAD et coll (1969) (Fig 2). The two indices are lateral ventricle (cella media) width temporal horn width (LV/TH) and third ventricle width mean temporal horn width (TV/TH). Quotients below 3.0 and 1.8 respectively indicate hydrocephalus. SJAASTAD et coll have chosen 1.7 as the border line value for the latter index. The points of measurement are not clearly defined in their work. The present authors have used for measurement a projection in the supine position with both temporal

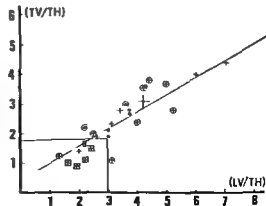


Fig 2 Correlation between cisternography (ventricular activity or not) temporal horn indices (lateral ventricle/ventricular activity index) and result of shunting procedure. Patients shunted before cisternography excluded. ● No ventricular activity + Ventricular activity ○ Shunted not improved □ Shunted improved. Bottom left: Border lines of temporal horn indices. Equation:  $TV/TH = 0.59 LV/TH + 0.39$ . Coefficient of linear correlation = 0.93.



Fig 3 Measured distances in determining temporal horn indices 1 = left ventricle 2 = third ventricle 3 = temporal horn

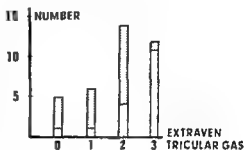
horns and lateral ventricles gas filled the beam being parallel to the long axis of the temporal horns. The measurement is taken on the transition between the lateral and the supracornual cleft according to Fig 3 which also indicates how the so-called septum — caudatum distance perpendicular to the head of the caudate nucleus is obtained, the third ventricle width is measured in the mid posterior part.

The hydrocephalic index is not correlated to the operative result (improved, mean index 0.40 not improved mean index 0.41) but to the result of cisternography as patients with ventricular activity have higher indices (mean index 0.41) than those without (mean index 0.34).

The discrepancy between the indices of the anterior and temporal horns when evaluating treatable hydrocephalus is possibly explained by the fact that the anterior horns yield earlier and more easily to the hydrocephalic process than the temporal horns. This is probably due to the different properties of the surrounding brain tissue. Geschwind (1968) has demonstrated that differential expansion depends on the structural properties of the walls of the container, that is the ventricular walls. Account must also be taken of the fact that the anterior horns are a common site of central atrophic conditions which may further confuse matters and make the index of these horns less correlated to hydrocephalic conditions.

Intraventricular absorption of cerebrospinal fluid is possible (SWEET et coll 1953, 1954, DAVSON 1967) which is also reflected in the cisternograms. This

Fig. 4 Correlation between the finding at cisternography and gas filling of extraventricular fluid pathways during encephalography. 0 Complete block over convexity. 1 C is filling of single furrows usually at interhemispheric fissure. 2 C is filling of Sylvian fissure but not parasagittally. 3 Normal gas filling of extraventricular fluid pathways.  $\square$  Ventricular activity.  $\square$  No ventricular activity.



reorption being to a certain extent proportional to the surface of the enlarged ventricular system (CRFITZ 1969). As long as the hydrocephalic state is only moderate a slight dilatation of the ventricular system manifesting itself mainly as a widening of the anterior horns, is probably sufficient to increase the ventricular surface and the absorption to achieve cerebrospinal fluid equilibrium (KILCORP et al. 1969). The subarachnoid block and resulting hydrocephalus thus seem to be compensated or arrested by ventricular dilatation and absorption: no benefit is then to be expected by ventricular shunting. When ventricular absorption is insufficient to compensate for the disturbance of the CSF circulation this incompenation seems to be accompanied by more marked dilatation of the temporal horns with indices below 3.0 (against the lateral ventricles) and 1.8 (against the third ventricle), respectively: as improvement by ventricular shunting occurs in a high percentage of these patients.

There was a high correlation between the degree of extraventricular gas filling and the result of the cisternographic investigation (Fig. 4). Half of the six patients improved by shunting had a complete block over the convexity and the other half no gas filling parasagittally. All those shunted without improvement had some gas filling of the Sylvian fissures. This is in comparison with those without ventricular activity, only one of 17 of whom had no gas filling over the convexity.

*Carotid angiography* was performed for investigating the source of intracranial hemorrhage in about two-thirds of the patients (36/51). The circulation time ad modum CRFITZ (1956) in subarachnoid hemorrhage is taken from angiographies performed more than three weeks after actual bleeding. Five of the 6 patients who were improved by shunting were subjected to angiography and their mean circulation time of 5.6 s did not differ significantly from that of other patients not improved or not shunted but with ventricular activity, with a circulation time of 5.4 s (6 and 14 patients respectively). Mean circulation time for those without ventricular activity at cisternography was 4.2 s (12 patients).

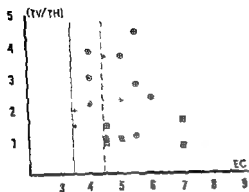


Fig 5 Correlation between cisternography cerebral circulation time temporal horn index (TV/TH) and result of shunt operation — — — 2 SD above normal circulation time ( $3.5 \pm 0.5$  s). Patients shunted before cisternography excluded ● No ventricular activity + Ventricular activity ○ Shunted not improved □ Shunted improved

Carotid angiography was performed in 3 of the present 5 patients with proven ectasia of the basilar artery. All vertebral circulation times were prolonged. Carotid circulation times were 5.5, 6.0 and 7.0 s. Two were shunted before cisternography and cannot be properly judged; of the remaining 3 patients all had ventricular activity during cisternography and were also treated with shunts. Only the patient with a circulation time of 7.0 s improved slightly and was the only one with a temporal horn index indicating hydrocephalus.

### Conclusion

The technique of ventricular shunting (Spitz-Holter medium valve) and method of judging the patients now described demand that the following criteria be fulfilled if improvement is to be expected in patients with a dilated, non-obstructed ventricular system: (1) Ventricular activity should be present during cisternography; (2) Temporal horns should be dilated as indicated by temporal horn indices (cella media width/temporal horn width  $< 3.0$ , third ventricle width/mean temporal horn width  $< 1.8$ ); (3) Circulation time determined by carotid angiography should exceed the normal value by 2 SD according to the GREITZ method (Fig 5); (4) Gas fillus, of the extracerebral fluid pathways, should be absent completely or at least over the hemispheres.

If the encephalography be performed correctly, appearances of hydrocephalus with absence of air over the convexities and frequently only partial filling of the basal cisterns must be taken as strongly indicative of blockage of the corresponding parts of the subarachnoid space. This has been evident in a material with so-called congenital communicating hydrocephalus (GRANHOLM & RADBERG 1963) and holds true for adult hydrocephalus.

It is practical when determining these diagnostic criteria to start with isotope cisternography. With no ventricular activity registered, severe disturbance of the



cerebrospinal fluid circulation may be excluded. Ventricular activity will usually be an indication for encephalography as well. Significant dilatation of the temporal horns and deficient gas filling of the extracerebral fluid pathways will suggest a shunt operation.

Carotid angiography to determine the circulation time and other possible lesions is of value in border line cases.  $^{133}\text{Xe}$  clearance estimation may also be used as this method is more ensue for revealing circulatory pathology than the determination of the circulation time by carotid angiography in normal pressure hydrocephalus (GREITZ 1969).

## SUMMARY

Criteria for successful shunting procedures particularly in normal pressure hydrocephalus based on a material of 51 patients with probable obstruction of the cerebrospinal fluid circulation are presented. The importance of dilatation of the temporal horns apart from ventricular isotope filling is stressed and the validity of other neuroradiologic parameters is also discussed.

## ZUSAMMENFASSUNG

An einem Material von 51 Fällen mit wahrscheinlicher Behinderung des Abflusses der Cerebrospinalflüssigkeit ohne Hypertension im Hydrocephalus wurden die Indikationen für eine Umdrainierung kritisch überprüft. Die Bedeutung einer Erweiterung der Temporalhörner abgesehen von einer Füllung der Hauptventrikel mit Isotopen wird betont und der Einfluss von anderen neuroradiologischen Faktoren besprochen.

## RÉSUMÉ

En se basant sur une série de 51 malades atteints d'obstruction probable de la circulation du liquide céphalo rachidien les auteurs présentent des critères permettant d'espérer un bon résultat des techniques de dérivation en particulier dans l'hydrocéphalie à pression normale. Outre la pénétration des isotopes dans les ventricules les auteurs insistent sur l'importance de la dilatation des cornes temporales et examinent la validité d'autres paramètres neuro radiologiques.

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## RECORDING OF THE LUMBAR CSF PRESSURE DURING ENCEPHALOGRAPHY IN HYDROCEPHALUS AND CEREBRAL ATROPHY

by

ARNE GREPE

Earlier investigations into the regulation of cerebrospinal fluid pressure during encephalography (AZAMBUJA et coll 1963 CROVQVIST et coll 1963) have indicated that injection of air into the CSF compartment without previous removal of fluid produces transient hypertension the height and duration of which is dependent on the volume injected. It has further been demonstrated that in patients with an intracranial process with or without intracranial hypertension the injection provokes a striking rise in pressure and prolongation of the time required before it returns to normal. The injection of large amounts of air may cause coma and convulsions should the pressure rise to levels higher than the diastolic arterial pressure.

The present investigation was initiated by observations that patients with a clinical history of communicating hydrocephalus and having typical encephalographic signs had a tendency to attacks of unconsciousness associated with a rise in arterial blood pressure during the procedure. A continuous recording of the lumbar CSF pressure during encephalography was therefore made in order to establish whether these patients had an inferior capacity of compensation for a rise in intrathecal pressure. This procedure would then constitute a means of

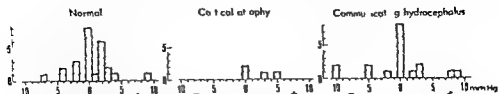


Fig. 1. Distribution of the initial CSF pressure (mm Hg) recorded at the level of the external meatus in examinations performed with the patient sitting.

differentiating cerebral atrophy and possibly of selecting patients suitable for operation.

**Material and Methods** The material is selected and includes mainly patients in whom the clinical findings made the diagnosis of low pressure hydrocephalus probable. In order to obtain an idea of the normal pressure regulating capacity, pressure recordings were also made in those in whom normal encephalographic findings were expected. However, as the encephalographic signs were unknown before the procedure, even this selected material is heterogeneous when classified according to the recognized appearances. The whole material comprises 47 patients divided into three well defined groups for analysis. The first group comprised encephalographically normal subjects without any notable changes in the ventricular system, cerebral fissures or sulci. Ventricular size was estimated by the LINDGREN method as the ratio between the maximal frontal horn width and the maximal internal skull width and an index of 0.30 was initially considered as the upper limit of the normal. This low value was deliberately chosen in order to make sure that no patients with ventricular dilatation were included. However, 4 patients with completely normal encephalographic appearances except for an index of 0.31 were found in this material. These were included among normals, especially as they presented no symptoms of disturbance in the CSF circulation. The inclusion of these patients failed to change the results from a statistical analysis. A second group contained patients who were considered as typical in that they had cortical atrophy with no changes in the ventricular system or the cerebral fissures but with pathologically widened sulci over the convexity including the parietal area. The third group was composed of patients with communicating hydrocephalus. The majority of these patients had encephalographic signs of convexity block hydrocephalus (GREITZ & GREPE 1971); these consisted of a widened ventricular system, normal or more often widened cerebral fissures and no air filling of the sulci over the convexity in spite of repeated attempts to fill them during the examination. One patient had an incisural block at encephalography. Twenty-five were normal, 4 had cortical atrophy and 18 patients had communicating hydrocephalus. The intrathecal

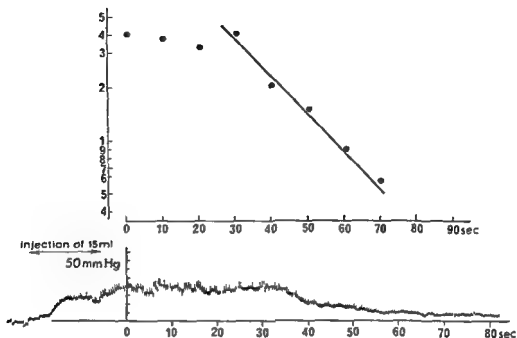


Fig 2 Lumbar CSF pressure curve in a normal subject following the injection of 15 ml of air. Curve plotted with a 10-second interval into a semilogarithmic system.

pressure was recorded by electromanometric methods by means of a needle introduced into the subarachnoid compartment between L3 and L4. This was connected via a polythene catheter to a pressure recorder linked to an ink recording oscillograph. The patient sat with the head held slightly forwards with a head clamp. All examinations were performed without general anaesthesia as this may change the intracranial pressure (GORDON & GREITZ 1970). The pressure recorder was positioned at the level of the external meatus, at which level the pressure is about 0. In the present material it varied between  $-10$  and  $+5$  mm Hg and only in 3 patients (1 normal and 2 with communicating hydrocephalus) was it above 5 mm Hg (Fig 1). Calibration was always performed before and after the pressure recording. A second needle was introduced between L4 and L5 for the air injection. A standardized injection technique was usually employed both as regards the amount of air injected and the time interval between the injections. Five milliliters of air were injected three times at 5 min intervals followed after a further 5 min by another 15 ml of air. Depending on the patient's reaction another 15 ml were sometimes injected after an interval of 10 min. The blood pressure was recorded before and after the injection. As this procedure is rather time consuming and as a pre-

Table

CSF pressure changes related to encephalographic findings. Ventricular index estimated by the LINDGREN method as the ratio between the maximal frontal horn width and maximal internal skull width. CSF pressure measured at external meatus in sitting position.  $T(1/2)$  denotes the time required for the CSF pressure to decrease to the half of any given value as estimated by graphical analysis of the monoexponential curve of the pressure decrease.

	Encephalographic diagnosis		
	Normal	Cortical atrophy	Communicating hydrocephalus
Number of cases	25	4	III
Age (years)			
Mean	40	60	58
Range	20-65	55-63	5-69
Ventricular index			
Mean	0.28	0.26	0.36
Range	0.24-0.31	0.23-0.29	0.31-0.49
Initial CSF pressure (mm Hg)			
Mean	0	7.9	0
Range	-7-+9	0-+5	-10-+9
Maximal rise in CSF pressure (mm Hg)			
Mean	42	53	53
Range	18-69	42-61	17-86
$T(1/2)$ (seconds)			
Mean	23	35	40
Range	14-40	27-48	13-80

luminary analysis after the first 30 pressure recordings indicated that several small injections failed to afford more than one or two injections the technique was later changed and an initial 5 ml injection was followed by a second administration of 15 ml after 5 min. The first injection of 5 ml served to exclude tonsillar herniation. The larger amount of 15 ml proved to give more informative pressure curves. It was found suitable to plot the curves with a 10 sec interval in a semilogarithmic system.

### Results

A typical pressure curve in a normal subject appears in Fig. 2. During the injection the pressure rises successively and after reaching a maximum level remains essentially unchanged and then falls to the original level within 2 min. These findings are in agreement with those of AZARUJA *et coll.* (1963).

The height of the maximum initial pressure rise for a given amount of air injected is likely to depend on the total volume of the subarachnoid space and

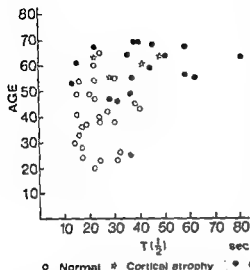


Fig 3 Relation ship between age and pressure regulating capacity as expressed by the time needed for the pressure to decrease from any given level to half this value during the fall  $T(1/2)$  denotes this half value

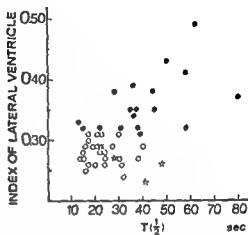


Fig 4 Relationship between the width of the ventricular system expressed by the LINDQUIST index and the pressure regulating capacity expressed as the half value —  $T(1/2)$  — for the fall

the elasticity of its walls. The pressure increase was not so marked in normal subjects as in patients with cortical atrophy or communicating hydrocephalus. In the latter two groups it was on an average at about the same level (Table). The period during which the pressure remained at the maximum level bore no correlation to age, encephalographic signs or the pressure regulating capacity as defined below. On the contrary during the same examination this period could be extremely variable with the same amount of air injected. When plotting the pressure changes in a semilogarithmic system it was evident that the decrease in pressure almost invariably followed a monoexponential curve (Fig. 2) the slope of which was more or less constant not only at different injections with different amounts of air but also at repeat encephalography including pressure recording at a later date. This slope may serve as an expression of the ability to compensate for the hypertension produced and the time needed for the pressure to decrease from any given level to half this value gives an expression  $T(1/2)$  of the pressure regulating capacity. Patients with normal findings at encephalography had a  $T(1/2)$  value varying between 14 and 10 sec with a mean of 23.1 sec the SD being 7.13 sec. The normal values are between 13 and 41.8 sec with 95% probability at a 95% confidence interval. The fairly large standard deviation is however, to a large extent due to the

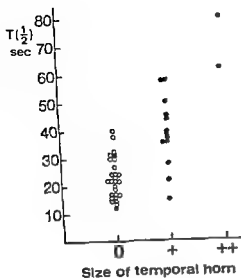


Fig 5 Relationship between the half value for the pressure fall —  $T(1/2)$  — and the size of the temporal horn in normal subjects O and patients with communicating hydrocephalus ● O denotes normal size of temporal horn + slight dilatation of temporal horn and ++ marked dilatation of temporal horn

relatively small number of patients examined ( $n=23$ ) and will probably be reduced in a larger material. There are certain indications that a  $T(1/2)$  value of 42 sec is too high an upper limit of normal. Four patients had a  $T(1/2)$  value above 30 sec; of these the patient with the highest recorded  $T(1/2)$  value (40 sec) had a pathologically slow CSF circulation at RIHSA cisternography. No RIHSA cisternography was performed in the other three patients. No correlation between age and the  $T(1/2)$  value appears to exist in patients without dilatation of the ventricular system (i.e. normal subjects and patients with cortical atrophy) (Fig 3). A highly significant correlation however exists between the width of the ventricular system as expressed by the ventricular index (i.e. the greatest width of the anterior horns related to the maximal internal skull width LINDGREN 1951) and the  $T(1/2)$  value when both normal subjects and patients with communicating hydrocephalus are considered together (Fig 4). There might also be correlation between the width of the temporal horn and the  $T(1/2)$  value in the latter group of patients (Fig 5).

### Discussion

The pressure rise following air injection is probably rapidly transmitted to all parts of a hydromechanical system such as the CSF compartment. The shape of the intrathecal pressure curve following air injection depends on many factors. Its height will largely be fixed by the amount of air injected, by the volume of the subarachnoid space and by the elasticity of its walls. The volume capacity



## QUANTITATIVE SCINTIGRAPHIC METHOD OF ESTIMATING THE CIRCULATION OF CEREBROSPINAL FLUID

by

ULLA LYNE LUNDEL and B. SÖDERBORG

The results of the investigation of the dynamics of cerebrospinal fluid (CSF) flow with isotopes are generally presented as a series of scintigrams recorded at different times and projections: the times are essential for the interpretation. Quantitative estimations of CSF flow with isotope techniques and external measurements have been performed to date only after intraventricular injection (MUSMAYER *et coll.* 1963; BERTI *et coll.* 1968), this method however involves a certain risk to the patient and physiologic conditions will be influenced by injection directly into the ventricles (SWIFT *et coll.* 1954). The aim was to develop a safe and reliable method of describing the dynamics of CSF flow quantitatively. Lumbar injection of  $^{125}\text{I}$  labelled human serum albumin (RISA) was therefore used and external measurements performed: the circulation of the RISA reflected that of the CSF (SWIFT *et coll.*)

*Technique.* The thyroid gland was blocked by administering 15 drops of iodine orally twice a day during a period of 10 days: this was begun three days before the examination when 100  $\mu\text{Ci}$  of RISA were injected intrathecally in the lumbar region with a fine needle (OD 0.6 mm). The volume was about 0.5 ml and the amount of protein generally about 1 mg, and always less than

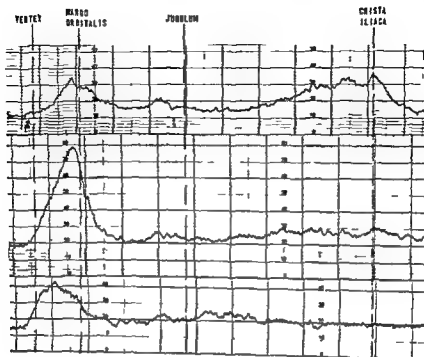


Fig 1 Linear scans at 3 24 and 48 hours in which the activity moves towards the cranium

4 mg the quantity that has been recommended as a maximum (Di Chiro et coll 1968) An amount of 0.5 ml CSF was aspirated into the syringe and injected with the isotope The needle was then flushed with 0.5 ml physiologic saline the patients were allowed to move about after the injection

The patients were examined with both profile curves (linear scans) and scintigrams for the next 3 to 5 days A slightly modified whole body scanner with a 3 inch crystal (Type SC 2 Nukab, Gothenburg) was used The pulse height analyzer was set for energies between 310 and 410 keV the pulses accepted are defined as counts The profile curves were obtained with the patients supine and the recordings made from the site of injection towards the cranium (Fig 1) A slit collimator (8 mm wide) was employed the detector speed being 2 mm per second An 11 hole focussed collimator was used for the scintigraphy The 90% response curve in air ranged from 5 to 9.5 cm from the collimator surface its maximum diameter being 0.6 cm The detector speed was 0.4 cm/s and the line spacing about 0.4 cm The low frequency suppression was governed by a rate meter The cut off setting was 900 cpm and the time



Fig. 2. Frontal view scintigrams with the areas of measurement.

constant  $0.1 \pm 1.5$  counts/0.1 s. The lowest of the dot factors 1, 2, 4 and 8 that made dot counting possible was selected. The ordinary projection used in pneumoencephalography was chosen for the frontal views. A lateral view scan was obtained when reflux of the activity into the ventricles was considered probable. The head was held in a plexiglass holder and anatomic landmarks, mainly the supra-orbital margin, the vertex and the symmetric midline, were marked in the scintigrams. The scanning time was sometimes shortened by restricting the scintigrams to the area of interest.

**Scintigrams.** A  $2 \text{ cm} \times 5 \text{ cm}$  frame was used for the measurements, corresponding to five scanning lines each with a length of 5 cm. Two areas were examined in the frontal projection: one in the region of the basal cisterns, the other over the paraventricular area (Fig. 2). These areas were marked in each of the scintigrams beginning with the one that displayed maximal activity in the region of the basal cisterns. A similar procedure was used for the paraventricular area.

The number of dots was counted in the frame, the activity is proportional to the number of counts although not to the number of dots. Low frequency suppression, if used, and the dead time of the printer influence the linearity between the counts and dots (TUNFILL & SODERBORG 1972). The relationship between counts and dots for different pulse rates and for usable dot factors

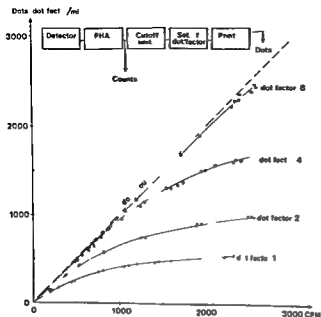


Fig 3 Correction curves between counts and dots without low frequency suppression

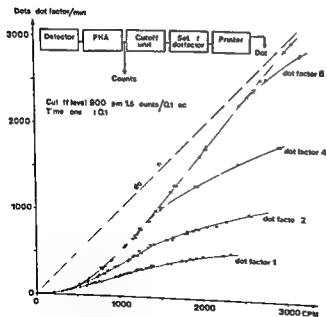


Fig 4 Correction curves between counts and dots with low frequency suppression

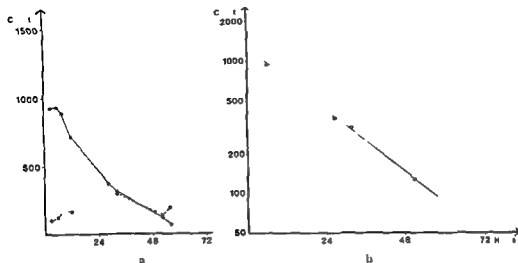


Fig 5 Concentration of RISA in relation to time a) Linear curves — Basal cisterns parasagittal area b) Semi logarithmic curve from the region of the basal cisterns

was determined experimentally (Figs 3, 4), the remaining parameters being kept constant. The counts were recorded with a fast scaler and the dots were registered simultaneously by means of an electronic switch attached to the printer and constructed so as not to affect the dead time of the printer. This was controlled by counting dots on paper that was moved by hand. The photons were obtained from a radioactive source during the calibration and thus appeared at random. The room background, ( $R_0$ ), was determined by recording counts with a scaler for at least ten minutes. This value was subtracted from the number of counts calculated and finally a correction was made for the isotope decay.

The diagram in Fig 5a depicts the counts corrected in relation to time from the two areas. The values from the basal cisterns for the same patient appear in the semi logarithmic diagram of Fig 5b. After the maximum was reached straight lines were obtained so that a biologic half life could be determined. No such regularity was evident in the parasagittal area. The whole procedure is illustrated in Fig 6. The counts in Figs 5 and 8 to 11 are corrected for decay.

*Control of the method* The functional stability (the setting accuracy) of the electronic equipment was checked by measuring the radiation from  $^{131}\text{I}$  sources. The measurements of the deviation from the theoretic decay curve of

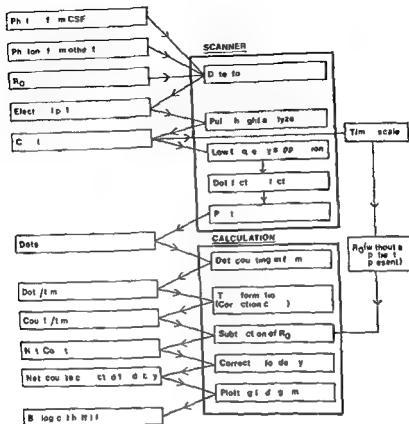


Fig. 6 Schematic presentation of the procedure

$^{131}\text{I}$  gave a deviation of 3 % during periods of 3 to 5 days. The measurement times for 10 000 counts were determined (Table).

The long term stability was checked by counting the room background. These measurements were made during 10 minute periods. The results of 64 measurements performed over a 2.5 year period gave a background of 208 counts/frame ( $\text{SD} = 12$  counts/frame or 6 %) corresponding to about 12 dots/frame ( $\text{SD} = 2$  dots/frame or 16 %).

The larger standard deviation for dots indicates that direct registration of counts with a timer-calculator is preferable for low count rates. The standard deviation of the background includes the statistical standard deviation and the true variations of the background, a mean value for background count,  $R_0$  was used for each patient.  $R_0$  consisting of natural radiation from the laboratory room, contamination, cosmic radiation and electronic noise.

**Table**  
*Methodologic errors*

Errors	Standard deviations	Standard deviations corresponding to	
		1 000 net counts	400 net counts
<i>Known random errors</i>			
Functional stability	3 of net counts	30	12
Determination of $R_0$	2.2 of $R$	5	5
Statistical SD	Square root of total count	35	25
Detector speed	1.3 of total counts	16	8
Conversion of dots to counts		4	4
Inhomogeneous uptake	2 of net counts	20	■
Decay constant (0.5 hours)	0.19 of net counts	2	1
Total known SD		53	31
Total known SD corrected for decay 24 and 48 hours after injection resp		58	37
<i>Unknown random errors</i>			
Variation in geometry of measurements mainly depending upon movements of the patient Scattered radiation			
<i>Observed random errors</i>			
Residual SD from straight lines for $^{229}$ scintigrams = 49			
<i>Systematic errors</i>			
Inhomogeneous uptake	Deviation $\pm 1$ of net counts	$\pm 10$	$\pm 4$
Radiation from tissue background	Deviation	$\pm 30$	$\pm 50^*$
<i>Minor errors regarding determination of biologic half life</i>			
Remaining activity in the syringes	12 of the activity		
Remaining activity at the site of injection	not calculated		
Long term stability	6 of total counts		

\* estimated values 24 and 48 hours after injection respectively

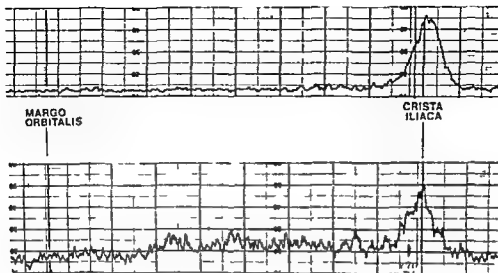


Fig 7 Linear scans at 4 and 24 hours with the activity trapped at the site of injection

The detector speed was tested about once a week. The calculated scanning time for a frame proved to be 62 s (observed deviation  $\pm 1$  s)

The injection procedure was simulated in a test tube as to control the activity remaining in the plastic syringe. The isotope was kept in the syringe for 10 min in 20 experiments and for 60 min in another 20 experiments. Just before emptying the syringe, 0.9% saline was drawn up in place of CSF, 12% (SD = 5%) of the activity remained in the syringe after emptying and no difference was observed between the two groups. The aspirated volume was varied in the test when 0.25 ml was aspirated the remaining activity was 19% (SD = 4%), 0.5 ml corresponded to 11% (SD = 2%) and 1 ml to 8% (SD = 2%). The amount of activity thus remaining diminished considerably when the volume of aspirated fluid was increased.

The activity generally moved from the site of injection to the cisterns and continued to the convexity. It was sometimes detected in the ventricular system. The profile curves were used principally to examine the initial distribution of the activity. These curves revealed when a sufficient amount was present in the cavity of the skull and when the first scintigram should be made. The injection failed in 11 out of 74 examinations (15%) which meant that the amount of activity reaching the skull was not sufficient for scanning. This occurred in spite of seemingly correct positioning of the needle through which the CSF flowed freely. In the profile curves the peak of activity at the site of injection



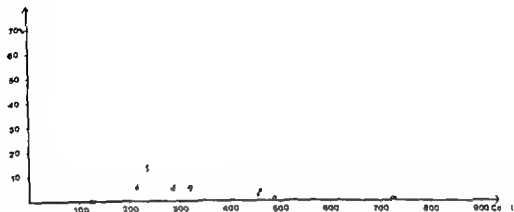


Fig 8 Percentage change in measured values obtained when moving the area of measurement one line in either direction O = positive X = negative change The dotted line indicates the sum of known random errors of importance in this test

sometimes remained symmetric indicating that a part of the isotope injected was trapped (Fig 7). No evidence of block was observed since at a further injection the activity travelled towards the skull.

It is essential that the patient does not move during the cinematography. This could be difficult to avoid even when using a head clamp. The position of the head was checked before and after the examination.

It is impossible to remeasure the same area in successive cinematograms with an accuracy greater than 1 scanning line i.e. 0.4 cm. The influence of moving the area of measurement was checked by displacing the frame one line in a cranial or caudal direction; this was done in 20 randomly selected cinematograms that had been used for determining the biologic half lives. The percentage change of measured values is given in Fig 8. The dotted line was derived from the sum of known random errors that were of importance during the time needed for scanning the area of interest. The errors were the statistical error, those from lateral inhomogeneity and from the estimation of  $R_0$  and finally rounding errors. Both the added and subtracted scanning lines were taken into account, resulting in two errors that were independent of each other. The difference between the measured and calculated errors indicates the degree of inhomogeneity near the original area; the difference is small (Fig 8).

The dot counting method presumes that the uptake in the area of interest is homogeneous i.e. that the variation in dot rate is only statistical. The homogeneity in the cranio-caudal direction was examined in the above mentioned test. In the orthogonal (lateral) direction in the cinematograms the homogeneity

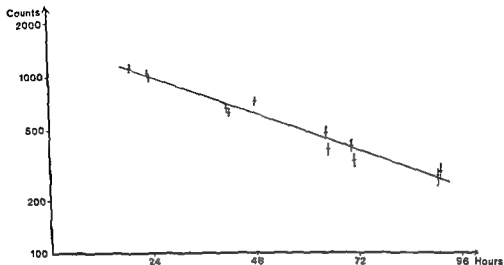


Fig 9 Errors of measurements due to uncertainty in time marked horizontally and the sum of all known random errors marked vertically

was tested by dividing the area of measurement into three equal parts one central and two lateral areas which were compared in 20 randomly selected scintigrams. The mean uptake in the central area was 14% (SD = 15%) higher than the average uptake in the whole area. The number of counts obtained from the area was compared to the summed up values of the three parts. The latter value was more accurate due to the homogeneity being greater in one third of the frame than in the whole frame. Assuming that this value was the correct one the analysis indicated that the value for the whole area was 1% (SD = 2%) too high. This degree of inhomogeneity seemed acceptable. The size of the area of interest was chosen as small as possible in order to give a homogeneous uptake and as large as possible to produce both satisfactory accuracy and make the refining of the area not too critical.

Frequent scintigrams were sometimes obtained with repositioning of the patient between each measurement in order to examine the precision of the single measurements. The errors of measurement from one examination marked in Fig 9 were the calculated sum of all known errors (Table). These calculated errors which were corrected for decay ranged from 32 to 58 counts the corresponding standard deviation being 12% and 5% respectively. The mean value was 41 counts. The residual standard deviation (derived from the deviations from the straight line) proved to be 49 counts. The residual standard deviation was also calculated from 229 scintigrams comprising 24 examinations each one

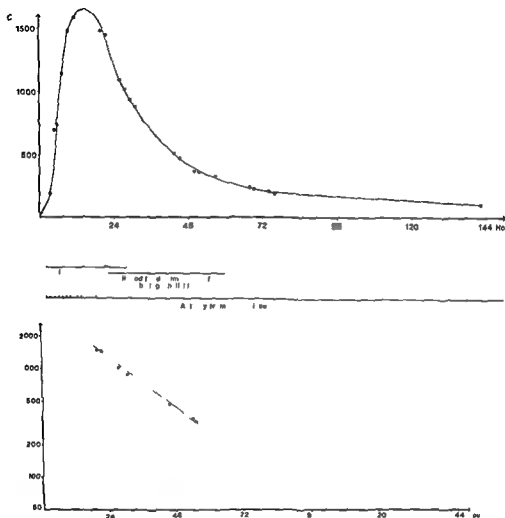


Fig 10 Measurements with a stationary detector. Above: Linear curve with schematic presentation of the different phases. Below: Semi logarithmic curve. Biologic half life is 15 hours.

having at least 5 usable values it proved to be 49 counts. Corresponding values for the 24 separate examinations ranged from 9 to 77 counts. Only rounding errors were accounted for from conversion of dots to counts. There was an additional experimental error that was not examined separately as it was included in the error from inhomogeneity.

The straight lines for determining the biologic half lives were drawn by hand. Consideration was paid to the relatively larger standard deviations in the lower

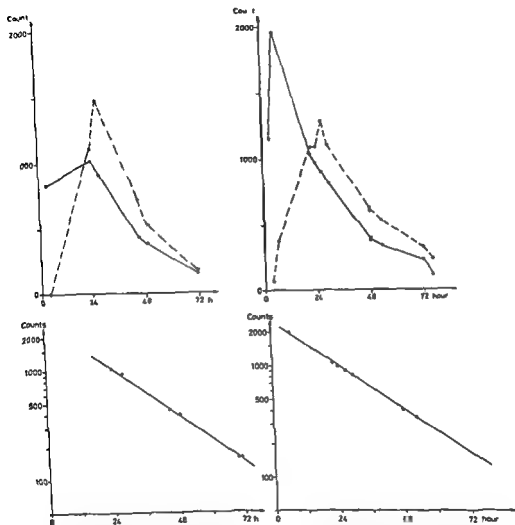


Fig 11 Two examinations of the same patient at a three-month interval. Above: Linear curves. Below: Semi logarithmic curves. Biologic half lives 17 hours (left) and 19 hours (right).

values and furthermore to the general form of the curves (Fig 10). The period for the determination of the biologic half life was estimated. When there are few available measurements values not belonging to the straight part of the curve may be included and result in too long a biologic half life. The length of the straight parts of the curves corresponded to about two days, after which the values generally became too low (in relation to the error) for measurements by the scintigraphic method.

The straight lines were analyzed (UCLA Biomedical Computer Program W J Dixon). A second degree polynomial regression analysis was performed for all 26 examinations having at least four usable values each. The analysis revealed that the quadratic term was not significant ( $p > 0.05$ ) except in two examinations. Linear regression was evident and the linear term was significant in all cases (in 19 out of 26 cases  $p < 0.001$ ). The values were not weighted in the analysis.

The biologic half life from the area of the basal cisterns did not seem to be dependent upon the amount of activity that reached the skull. This could be presumed when examining profile curves that sometimes disclosed activity remaining at the site of injection and was also supported by the results of replications of the whole examination in 3 cases, in 2 of these the total activities in the basal cisterns differed considerably between the two examinations. The relationship between the activities 24 hours after injection was 1.37 and 1.15, respectively. The differences regarding the biologic half lives were 3.11 and 13% in the three cases. One of these cases is represented in Fig. 11.

An analysis of the uncertainty in the determination of the biologic half life was made in 16 randomly selected cases by means of weighted linear regression (BROWNLEE 1960). The weights were chosen so as to be inversely proportional to the variance of the values of the corrected net counts (Table). The analysis indicated that the uncertainty in the determination of the biologic half life was 8%. The corresponding value with non weighted regression was 9.5%. The uncertainty in the determination of time was not considered in the regression analysis; it was less than 0.5 hours (Fig. 9).

### Discussion

In the basal cisterns the CSF from the spinal canal containing RISA becomes mixed to some extent with pure CSF from the ventricles; there are physiologic reasons to assume that mixing of CSF takes place in this region (OLDENDORF 1967, DE BOLLAY 1966) which therefore was selected for measurements. The results disclose that approximately straight lines were obtained from this area and support the assumption that satisfactory mixing takes place. However, it has not been established that mixing is complete; the accuracy of the method being limited. The results could also be compatible with a complicated multi-compartmental system.

Assuming that the activity is well mixed, the disappearance rate will be proportional to the amount of activity in the volume where the mixing takes place.

When no more activity is added, the disappearance will occur according to the function

$$A_t = A_0 e^{-\lambda t} \quad (1)$$

where  $A_0$  = activity at time 0  $A_t$  = activity at time  $t$   $\lambda$  = rate of outflow,  $V$  = the volume where the mixing takes place

If the variation of the activity is expressed by function (1), straight lines will be obtained in a semi logarithmic diagram, where the time is plotted along the linear axis and the activity along the logarithmic axis. Function (1) assumes that no activity is transported into the area. If activity is brought into the area according to a function  $a(t)$ , a more complicated formula is needed to describe the variation of the activity

$$A_t = \int_0^t a(s) e^{-\lambda(t-s)} ds \quad (2)$$

where  $s$  is a dummy variable  $0 \leq s \leq t$  and the activity at the time 0 is 0

The measurements reveal that a short time after  $A_t$  has reached its maximum straight lines are obtained in a semi logarithmic diagram indicating that the inflow of activity has become negligible. Formula (1) is then valid.

Only a fraction of the radiation from  $A_t$  is detected. This fraction is determined by the geometry of measurement and the detector system. The efficiency differs between various detector systems. The geometry of measurement will be constant for each case if the position of the patient be unchanged and if there is a homogeneous concentration of the isotope in the volume measured.

Activity in other tissues is also measured either as primary or as scattered radiation. The influence from the latter will be diminished with pulse height analysis and adequate collimation. Should the scattered radiation originate from the CSF near the measured area, the biologic half life will not be affected as the relative disappearance rate  $\frac{\lambda}{\lambda + \lambda'}$  would be about the same.

The activity in the CSF decreases gradually. As RISA is eliminated slowly from the body, the radiation from tissue background becomes increasingly important. Late scintigrams give an estimation of the contribution from other tissues. The curve in the semi logarithmic diagram is thus gradually flattened. The tissue background in the area of the basal cisterns is low, which is well known from brain scanning. Calculations were made in the area of interest from three brain scans obtained after the intravenous injection of RISA. It was established that the activity corrected for decay varied only slightly. The mean count rate

## FACTORS INVOLVED IN SURGICAL MANAGEMENT OF NORMAL PRESSURE HYDROCEPHALUS

by

W J MICHELSEN, E B SCHLESINGER and S BAILEY

The triad of dementia, gait difficulties and urinary incontinence in an aging patient are the main signs that bring to mind normal pressure hydrocephalus (HAKIM & ADAMS 1965; ADAMS et coll 1965). In this syndrome spinal fluid pressure is normal, and encephalography reveals hydrocephalus with little or no air passing the incisura. Isotope encephalography with RISA reveals ventricular filling with little or no isotope over the hemispheres (SCHLESINGER et coll 1968). The patients improve strikingly with cerebrospinal fluid shunts (OJEMAN et coll 1969).

Sixteen patients with this syndrome have been examined and treated from 1965 to 1970. Twelve of these improved and four did not improve. In this report the vagueness of the clinical signs will be emphasized and our criteria for successful therapy along with the complications will be described.

### Case reports

*Case 1* Male, aged 68. He was first seen in the neurology clinic in June 1960 after onset of weakness of both legs. His gait was broad based and hesitant. The right plantar response was extensor. There was no objective weakness. LP pressure: 80 mm water.



Fig 1 Case 1 Total interventricular concentration of isotope 48 hours after lumbar subarachnoid injection of RISA

**Diagnosis** akinetic Parkinson's disease The gait worsened over the next six years Slight memory loss and difficulty in abstracting occurred after 6 years Prostatic carcinoma was diagnosed and orchiectomy done By March 1967 he could not walk but could move about well in bed No grasp or snout His memory worsened and occasional incontinence occurred He was re-admitted to the hospital now aged 70 LP opening pressure 77 mm water protein 41 mg % Intrathecal injection of RISA was followed by delay of the isotope in the lumbar subarachnoid space and ventricular filling at 24 hours At 48 hours after injection isotope (Fig 1) was noted only in the ventricles this observation is made only in communicating hydrocephalus However encephalography (Fig 2) revealed an enlarged Sylvian fissure and callosal sulcus In spite of the findings a low pressure ventriculo-atrial shunt was placed

The patient improved remarkably Five weeks post-operatively he suddenly became hemiplegic and aphasic Craniotomy after angiography revealed a large subdural hygroma He slowly recovered and the shunt was not ligated 30 months post-operatively his gait was better than 14 years earlier and at age 78 he is caring for his invalid wife

**Case 2** Male aged 63 a former alcoholic with a history of two years gait difficulty He noted pain and weakness of his anterior thighs when walking Six months prior to admission he began to have difficulty with his balance There is a history of meningococcal meningitis 26 years ago General examination Marked emphysema abdominal mass with bruit and absent dorsalis pedis pulses Neurologic examination Patient oriented with fair fund of knowledge Gait was broad based shuffling and magnetic in nature No motor weakness no pathologic reflexes and no incontinence LP pressure 80 mm water with protein 29 mg % Femoral aortography revealed widespread vascular disease of the legs but not surgically correctable Cerebral angiography Widespread intra and extracranial vascular disease At 48 hours after intrathecal injection of RISA isotope was found solely within the ventricles Encephalography was consistent with cerebral atrophy A low pressure shunt was placed because of our experience with Case 1 His gait immediately improved and the leg pain disappeared Three months post-operatively his gait again deteriorated and the shunt was revised He did well until six months later when he became obtunded and his gait worsened A subdural hygroma was removed and he improved It is now two years since his first shunt and his gait has remained improved.





Fig. 2 Same case as in fig. 1. Encephalogram with air in enlarged callosal sulcus and Sylvian fissures.

**Case 3** A male aged 64 sustained a fracture of his left femur 5 years prior to admission. Subsequently his gait became shuffling and broad based. He began to fall frequently and lost the ability to have an erection. No pain or paresthesia noted. On admission except for a slightly shortened left leg general examination was normal. He was alert and oriented with no evidence of dementia. He was not incontinent. Gait was broad based, shuffling and magnetic in nature. Romberg was positive. Sensory examination was normal. The patient could not walk on all fours because his hands were all o'glued to the floor. No pathologic reflexes. I.P. pressure 180 mm water protein 27 mg%. Intrathecal injection of RIS\ revealed persistent ventricular filling over 48 hours without isotope movement out of the ventricles and encephalography revealed hydrocephalus with incisural block. A lumbar peritoneal shunt was placed and his gait improved remarkably. He is now back at his regular work with almost normal gait and reports having voluntary erections and sexual intercourse for the first time in six years.

**Case 4** A male aged 79 had progressive leg weakness over four years. On admission he was unable to walk. Recent onset of poor memory and no urinary incontinence. On examination he had a mild organic mental syndrome, increased tone in both legs with a positive Babinski. Positive snout and glabella reflexes. The strength of his legs in bed was almost normal in spite of which he could not walk. Sensory examination was normal. I.P. pressure 160 mm water protein 24 mg%. Intrathecal injection of RIS\ revealed prolonged ventricular filling but a small amount of isotope moved into the Sylvian fissure and over the convexity (Fig. 3) at 2 hours as well as 48 hours. Encephalography was consistent with cerebral atrophy. A low pressure ventriculo-atrial shunt was placed. The patient did not improve and fourteen months post-operatively died from a myocardial infarction. His neurological condition had not improved.

### Discussion

The 6 cases characterize a small number of patients at the Neurological Institute of New York who have had the diagnosis of normal pressure hydrocephalus

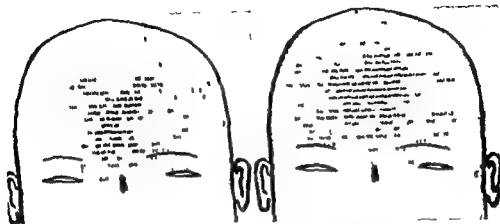


Fig 3 Case 4 Two hour (left) and 32 hour (right) intrathecal RISA a p scans The majority of the isotope is intraventricular on both scans but there is also marked activity in the subarachnoid pathways over the hemispheres

Their primary abnormality was a long standing gait disturbance. This had been diagnosed as akinetic Parkinson's disease in Case 1, intermittent claudication in Case 2, and correctly as hydrocephalus in Case 3 because of our earlier experience. Mental symptoms and incontinence were not present in 2 cases. The motor disturbance extended to the upper extremity in one case. Two of the patients who improved had ventricular dilatation without incisural block at encephalography.

The only examination that clearly differentiated the improved cases was the intrathecal RISA test. If the isotope remained solely within the ventricular system, cerebrospinal fluid shunting produced improvement. If there was egress of the isotope from the ventricles to the subarachnoid space over the convexity, no improvement occurred. Three other patients with slight movement of the isotope from the ventricles have been shunted with no improvement. This is in contrast with ten patients where the isotope concentrated entirely within the ventricles, all of whom improved.

Two of these patients developed subdural hygromas after shunting procedures. In both cases the hygroma was evacuated but only in the second case was the shunt temporarily ligated. This complication prompted the use of a lumbar peritoneal shunt in Case 3.

### Acknowledgements

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## SUMMARY

A group of patients with normal pressure hydrocephalus is presented. The major clinical finding was a long standing gait disturbance. The patients all underwent spinal fluid shunting procedures. Improvement was noted in those cases in which intrathecal RISA concentrated solely in the ventricular system over 48 hours. It is suggested that patients with long standing gait disturbances be investigated for this syndrome.

## ZUSAMMENFASSUNG

Es wird eine Gruppe von Patienten mit Hydrocephalus bei normalem Druck beschrieben. Der hauptsächliche klinische Befund war eine langanhaltende Gangstörung. Bei allen Patienten wurden Spinalflüssigkeits Shunt Eingriffe vorgenommen. Verbesserung wurde in solchen Fällen gefunden, bei denen intrathecal gegebenes RISA lediglich im Ventrikel system über 48 Stunden konzentriert war. Es wird vorgeschlagen, Patienten mit langanhaltenden Gangstörungen auf dieses Syndrom hin zu untersuchen.

## RÉSUMÉ

Les auteurs présentent un groupe de malades atteints d'hydrocéphalie à pression normale. Le signe clinique principal est un trouble de la marche datant de longtemps. Tous ces malades ont subi une dérivation du liquide céphalo-rachidien. Les auteurs ont constaté une amélioration dans les cas où le sérum albumine iodée radio active injectée dans l'espace sous-rachnoïdien se concentrait uniquement dans le système ventriculaire pendant 48 heures. Ils pensent que les malades qui présentent des troubles de la marche datant de longtemps devraient être examinés pour rechercher ce syndrome.

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## SCINTIGRAPHY OF THE NORMAL PERICEREBRAL FLUID CIRCULATION

by

R. OSERSON and T. MARTINI

Radiotopic investigations may provide a good indication of the morphology of the subarachnoid space, an appreciation of the movements of the cerebrospinal fluid (direction, speed and fate) and an approximation of the absorption sites and rate of tagged proteins.

*Material and Methods* More than 200 normal isotope cisternograms were reviewed. Scintigrams were recorded with a conventional rectilinear dual scanner 1 to 2, 5 to 6 and 24 hours after the suboccipital cisternal injection of 150  $\mu$ Ci RISA. Criteria of normality have been gradually worked out in aged subjects, average adults and children from two main points of view: cisternal morphology and fluid dynamics. Both are often so closely involved that their separation is almost impossible.

### Results

*Anatomy* The CSF contaminated at the cisterna cerebellomedullaris spreads through the pericerebral cisterns through several interconnected pathways, the main direction being medial from the region of the foramen of Magendie to the interhemispheric sulci.

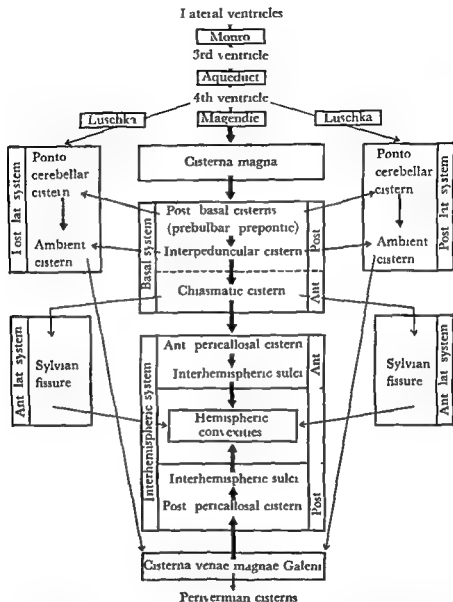


Fig. 1 Diagrammatic representation of the pericerebral circulation in the subarachnoid space

(1) The basal system CSF from the foramen of Magendie leaves the area of contamination progressively to reach the posterior basal cisterns (prebulbar preponic interpeduncular chiasmatic and pericarotid)

(2) The superior system This is interhemispheric and spreads out anteriorly from the chiasmatic cistern to follow the pericallosal arteries and their branches



Fig 2 Right lateral scintigram of the head a) 50 b) 90 and c) 120 min after suboccipital cisternal injection of 150  $\mu$ Ci RISA. Basal cisterns Cisterna cerebellomedullaris (1) ambient system (2) Sylvian system (3) cervical perimedullary subarachnoid space (○) prebulbar and preponic cisterns (→) external auditory meatus (●)



Fig 3 Normal radioisotope cisternography a) Lateral view 5 h and 30 min after cisternal injection b) Anterior view at 11 h The bulk flows through the superior medial system pericallosal (→) in (a) interhemispheric in (b) where the Sylvian fissures (↔) are poorly contaminated

Posteriorly it starts at the cisterna venae magnae Galeni and follows the medial branches of the posterior cerebral arteries for the posterior part of the brain and the pericallosal sulci for the middle part of the brain

(3) Two lateral systems of pathways complete the drainage. The posterior lateral system (ambient system) carries the CSF from the pontocerebellar cisterns (in connection with the foramina of Luschka) to the cisterna venae magnae Galeni and passes through the ambient cisterns. The anterior lateral system (Sylvian system) extends from the chiasmatic cistern to the hemispheric convexities through the Sylvian fissures.

This schematic representation is in accordance with the pericerebral morphology and with the disturbances of pericerebral fluid dynamics reported (MARTIN & OBERSON 1969). It underlines the prominent role of the basal system. The lateral systems are paired i.e. complementary.

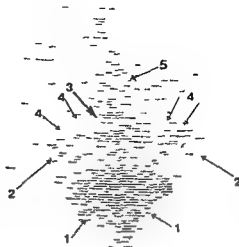


Fig. 4. Normal radioisotope cisternography. Posterior view at 1 h. Cisterna magna (1), pontocerebellar cisterns (2), cisterna venae cavae magna (3), ambient cisterns (4), posterior part of medial system (5).

With these patterns in mind it is easier to read the scintigrams. A general survey of the pericerebral circulation (the third circulation) is represented in Fig. 1.

**Scintigraphy.** The pericerebral fluid circulation is considered normal when the movement of the CSF in the subarachnoid space is free. Successive scintigraphies establish these facts. Normal patterns are easy to recognize (Figs 2 to 5).

The liquid contaminated in the cisterna magna exhibits a pericerebral diffusion: there is no apparent ventricular contamination. The position of the patient or of his head has no influence on the centrifugal distribution. The suboccipital cisternal injection is preferred because it is more selective (better contrast in the cranial cavity, less irradiation of the spinal cord). Siphoning and drawing off of fluid should be avoided. Diffusion and resorption of the pericerebral contaminated liquid are regularly progressive throughout the whole subarachnoid system. The last tagged proteins — probably the heaviest — are observed at 24 hours on the frontoparietal hemispheric convexities in adults, this occurs at 12 hours in children. Frontal views — anterior or posterior — present the various cisterns symmetrically. Normal and pathologic scintigraphic appearances of pericerebral fluid circulation have already been described (DI CUNEO 1966, ALKER & LESLIE 1969, KILGROE et al. 1969, MARTINI & OBERSON 1969, OBERSON & MARTINI 1968, OBERSON 1970). Certain basic details however deserve special attention.

The basal cisternal system is demonstrated in successive lateral scintigrams of the head (Fig. 2). The major branch lines of diffusion appear at 2 hours.



Fig 5 Pathologic radioisotope cisternographies. Anterior view of two patients with brain cortex atrophy (hydrocephalus ex vacuo). Candlestick pattern of CSF distribution well demonstrated. Dilated Sylvian fissures (1) pericallosal and interhemispheric systems (2)

these are the ambient system and the Sylvian fissure. The cistern of the quadrigeminal plate is contaminated through the ambient cisterns supplying the superior medial system via the interhemispheric sulci. The importance of the inferior medial system, the prebulbar, preponic, interpeduncular and chiasmatic cisterns is recognized. As yet interest has not been focussed on the interhemispheric pathway in spite of BEDFORD'S demonstration of its primary significance. The interhemispheric drainage is often so large as to be misinterpreted as a ventricular contamination (Fig 3).

The posterolateral system of drainage is best seen in the posterior scan at between 3 and 7 hours following injection (Fig 4).

At the same time the anterolateral system is clearly demonstrated in the anterior scan. The candlestick appearances of the diffusion of the contaminated fluid are well known (Fig 5) but the midline drainage, although it is the largest, is often overlooked.

The crescent pattern of radioactive distribution at 24 hours in the anterior scan is often interpreted as indicating the area of protein resorption. Substantiates the claim that a pericerebral centrifugal fluid movement exists. It also proves that only the largest — or heaviest — particles reach the cerebral convexities not because it is their sole site of absorption but because they have the longest subarachnoid biologic half life.

## SUMMARY

Two hundred normal radioisotope cisternographies were reviewed. The morphology of the subarachnoid space, the movements of the cerebrospinal fluid and the site and rate of absorption of tagged proteins are considered in turn.



## ZUSAMMENFASSUNG

Zweihundert normale Cisternographien mit Radioisotopen werden besprochen. Die Morphologie des subarachnoidalen Raumes, die Bewegungen des Liquors und der Platz und die Geschwindigkeit der Absorption des gezeichneten Proteins werden jeweils betrachtet.

## RÉSUMÉ

Les auteurs présentent deux cents cisternographies radioisotopiques normales. Ils examinent la morphologie de l'espace sous arachnoïdien, les mouvements du liquide céphalo-rachidien et le site et la rapidité d'absorption des protéines marquées.

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## DETERMINATION ANGIOGRAPHIQUE DU CARACTERE DE L'HYDROCEPHALIE INTERNE ET DU NIVEAU EVENTUEL DE L'OBSTRUCTION

par

J PETROV

Le developpement contemporain de l'angiographie carotidienne rapide en serie a rendu possible l'etablissement du diagnostic angiographique de l'hydrocephalie interne dans tous les cas et ce grace aux donnees provenant des diverses phases et projections de l'angiographie carotidienne

Les alterations arterielles hydrocephaliques sont deroulement et aspect en arc tendu des arteres cerebrales anterieure et pericalleuse elevation et tension du groupe sylvien « en diagonale » (18) aplatissement et deformation en T de la bifurcation terminale du siphon carotidien (29), deplacement lateral du groupe sylvien (13) depression des segments proximaux de l'artere cerebrale posterieure (19) deplacement medial de la branche calcarine de l'artere cerebrale posterieure (15) acceleration de la vitesse de la circulation cerebrale surtout par allongement de la phase arterielle (26) parallelisme des segments terminaux de l'artere cerebrale anterieure et de l'artere cerebrale moyenne (5) deplacement lateral deformation et depression des arteres lenticulo-striees (2) opacification simultanee des deux arteres pericalleuses (31) tension generale et amincissement de toutes les arteres cerebrales (9)

Les altérations angiographiques veineuses en cas d'hydrocéphalie interne décrites jusqu'à présent s'expriment par une augmentation de la distance entre le sinus longitudinal inférieur et la veine cérébrale interne (20) opacification précoce du système veineux profond (30) aplatissement et dépression de la veine cérébrale interne et déplacement latéral de la veine thalamo-striée (12) allongement et tension de la veine du septum pellucidum et de la veine thalamo-striée ainsi que de leurs affluents (17) déformation en arc accompagnée de convexité latérale de la veine thalamo-striée (27) agrandissement de l'angle entre la veine thalamo-striée et la veine cérébrale interne sur le phlebogramme de face (22) prolongation de la période de l'opacification de toutes les phases de la circulation cérébrale (14) ouverture et arrondissement de l'angle veineux du cerveau (4-10) et agrandissement de l'angle entre le sinus longitudinal inférieur et la veine de Galien (11).

L'image angiographique de l'hydrocéphalie interne consécutive à l'obturation par un processus supratentorial dans le 3ème ventricule est presque identique à celle qui est provoquée par un processus expansif et adhésif dans la fosse crânienne postérieure ou par une atrophie cérébrale massive. L'opinion classique qui est encore en vigueur prétend qu'il est impossible de déterminer sur la base de données de l'angiographie carotidienne le caractère de l'hydrocéphalie interne de même qu'il est impossible de définir au quel niveau de l'occlusion en cas d'hydrocéphalies obstructives (6, 11, 15, 21, 28). Ce ne sont que des auteurs isolés qui font ressortir des critères basés sur le diagnostic angiographique pour la différenciation de l'hydrocéphalie. C'est ainsi que pour distinguer l'hydrocéphalie due à une tumeur dans le 3ème ventricule de celle qui est provoquée par des adhérences ou une tumeur dans la fosse crânienne postérieure, ACHARD (1962) attire l'attention sur le déplacement en avant du segment proximal inférieur de l'artère pericalluse, observé en cas de tumeurs dans le 3ème ventricule. Tandis que LOICREY (1956) décrit dans l'hydrocéphalie due à une tumeur du cervelet une élévation importante de l'artère cérébrale postérieure et une dépression ou un trajet normal de la même artère en cas de blocage de l'aqueduc de Sylvius. LEGER (1948) de son côté, constate en cas de tumeurs du cervelet un déplacement de la veine basale et de la veine de Galien en avant et en haut ou contraire. JOHANSSON (1951) trouve dans ces mêmes cas un déplacement de la partie postérieure de la veine basale en avant et en bas.

L'établissement de critères spécifiques et permanents provenant de l'angiographie carotidienne à l'aide desquels on pourrait définir le niveau de l'occlusion en cas d'hydrocéphalies obstructives ainsi que le caractère de celles-ci (obstructives ou communicantes) présente un grand intérêt au point de vue diagnostic différentiel, traitement ou pronostic. C'est pourquoi le principal objectif du présent travail consistait à examiner les altérations angiographiques

hydrocephaliques, pour établir de pareils critères. Evidemment il est question de tels critères sur la base desquels on puisse au moins affirmer s'il s'agit d'un niveau d'obstruction supra ou subtentorial.

*Matériel.* Nous avons choisi 120 malades présentant des symptômes d'hydrocephalie tout en les soumettant, dans un court intervalle de temps et presque parallèlement à une angiographie carotidienne et à une pneumographie.

Du point de vue appartenance nosologique il s'agissait de malades présentant diverses affections organiques systémiques du système nerveux central, ayant amené à une dilatation symétrique légère ou modérée du système ventriculaire (64) tumeurs dans le 3ème ventricule (28) et tumeurs ou adhérences dans la fosse crânienne postérieure (28).

D'après le degré de la dilatation ventriculaire les malades étaient répartis en trois groupes.

1 Dilatation ventriculaire légère	33
2 Dilatation ventriculaire modérée	31
3 Dilatation ventriculaire massive	56

Dans le premier et le second groupes il s'agissait de patients présentant une hydrocephalie communicante tandis que le troisième groupe comprenait exclusivement des malades souffrant d'hydrocephalie obstructive.

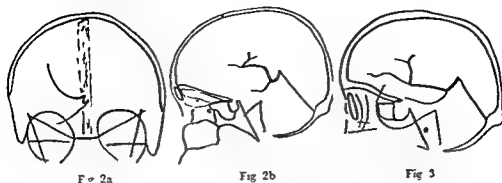
L'établissement du degré de la dilatation ventriculaire a été effectué à l'aide des indices pneumographiques d'EVANS (1942) et des indices phlébographiques (PETROV, 1969) établis par nous-mêmes à titre d'orientation, entre lesquels on remarque un parallélisme presque complet.

Les altérations artérielles chez les trois groupes de malades souffrant d'hydrocephalie dont il est question ci-dessus se résument sur l'angiogramme de face (Fig 1 a) en l'absence de déplacement de l'artère percalcaire par rapport à la ligne médiane à l'exception de 4 cas du 3ème groupe et ce lors de tumeurs dans le 3ème ventricule chez lesquelles il existait une légère asymétrie dans le degré de dilatation des deux ventricules latéraux. Il existait une légère déviation (de quelques mm) de l'artère percalcaire par rapport à la ligne médiane. Sur l'angiogramme de profil (Fig 1 b) les altérations hydrocephaliques artérielles (qui ne se distinguaient pas des descriptions classiques énoncées au début du présent travail) n'étaient nettes que chez les malades du 3ème groupe. Dans le 2ème groupe elles étaient seulement esquissées tandis que dans le 1er groupe elles manquaient. Il faut mentionner en outre que les modifications hydrocephaliques artérielles chez les malades du 3ème groupe étaient les mêmes tant pour les tumeurs du 3ème ventricule que pour les tumeurs et les adhérences dans la fosse crânienne postérieure.

Les alterations angiographiques veineuses en cas d'hydrocephalie interieure decrites jusqu'a present s'experiment par une augmentation de la distance entre le sinus longitudinal inferieur et la veine cerebrale interne (20) opacification precoce du systeme veineux profond (30), aplatissement et depression de la veine cerebrale interne et deplacement lateral de la veine thalamo-strie (12) allongement et tension de la veine du septum pellucidum et de la veine thalamo-strie, ainsi que de leurs affluents (17) deformation en arc accompagnee de convexite laterale de la veine thalamo-strie (27) agrandissement de l'angle entre la veine thalamo-strie et la veine cerebrale interne sur le phlebogramme de face (22) prolongation de la periode de l'opacification de toutes les phases de la circulation cerebrale (14) ouverture et arrondissement de l'angle veineux du cerveau (4-10) et agrandissement de l'angle entre le sinus longitudinal inferieur et la veine de Galien (11).

L'image angiographique de l'hydrocephalie interne consecutive a l'obturation par un processus supratentorial dans le 3eme ventricule est presque identique a celle qui est provoquee par un processus expansif et adhésif dans la fosse cranienne posterieure ou par une atrophie cerebrale massive. L'opinion classique, qui est encore en vigueur pretend qu'il est impossible de determiner sur la base de donnees de l'angiographie carotidienne le caractere de l'hydrocephalie interne de meme qu'il est impossible de definir au si le niveau de l'occlusion en cas d'hydrocephalies obstructives (6, 8, 15, 21, 28). Ce ne sont que des auteurs isolés qui sont ressortis des criteres bases sur le diagnostic angiographique pour la differenciation de l'hydrocephalie. C'est ainsi que pour distinguer l'hydrocephalie due a une tumeur dans le 3eme ventricule de celle qui est provoquee par des adherences ou une tumeur dans la fosse cranienne posterieure, ARVOLI (1962) attire l'attention sur le deplacement en avant du segment proximal inferieur de l'artere pericalléuse observe en cas de tumeurs dans le 3eme ventricule. Tandis que LOEFGRÉN (1956) decrit dans l'hydrocephalie due a une tumeur du cervelet une elevation importante de l'artere cerebrale posterieure et une depression ou un trajet normal de la meme artere en cas de blocage de l'aqueduc de Sylvius. ECKER (1948) de son cote constate en cas de tumeurs du cervelet un deplacement de la veine basale et de la veine de Galien en avant et en haut au contraire JOHANSSON (1954) trouve dans ces memes cas un deplacement de la partie posterieure de la veine basale en avant et en bas.

L'etablissement de criteres specifiques et permanents provenant de l'angiographie carotidienne a l'aide desquels on pourrait definir le niveau de l'occlusion en cas d'hydrocephalies obstructives, ainsi que le caractere de celles-ci (obstructives ou communicantes) preenterait un grand interet au point de vue diagnostique differentiel traitement ou pronostic. C'est pourquoi le principal objectif du present travail consistait a examiner les alterations angiographiques



F = 2a

Fig 2b

Fig 3

Fig 2 M me cas que fig 1 a) Schema d'un phlebogramme de face disposition de la veine cerebrale interne le long de la ligne mediane Déplacement lateral massif a convexite laterale de la veine thalamo-strie. b) Schema d'un phlebogramme de profil angle veineux leg rement souleve et ferme Segment anterieur de la veine cerebrale interne leg erement aplati et souleve Calibre irregulier de la m me veine

Fig 3 Tumeur dans le 4eme entricule avec obstruction de l'aqueduc de Sylvius. Schema d'un phlebogramme de profil an le veineux ouvert Depression du sommet de cet angle Aplatisse ment de la veine cerebrale interne accompagne d'une depression s multanee de cette veine

bien a une leg ere ouverture de l'angle veineux ans deplacement du ommet de celui-ci en haut et en bas ou bien en avant et en arriere (chez 8 malades) C est sur le phlebogramme de profil qu'on a pu etablir chez les malades du 3eme groupe les modifications les plus manifestes frequentes et permanentes Pourtant le caractere de ces modifications dependait dans une certaine mesure de la disposition de l'obstruction C est ainsi que lors de l'obstruction sus-entoriel (tumeur du 3eme ventricule) (Fig 2 b) chez 27 malades (sur un total de 20) presentant une telle localisation de la tumeur, le ommet de l'angle veineux etait souleve L'angle veineux lui meme etait nettement ouvert (chez 10 malades) ou bien ensiblement ferme (chez 5 malades) La veine cerebrale interne formait un arc anterieur aplati et en meme temps leg erement souleve (chez 14 malades) On a constate chez 3 malades un arc anterieur accentue sur la meme veine On a trouve chez 9 malades une leg ere deformation en marche d'escalier du trajet de la veine cerebrale interne ■ un calibre irregulier de celle ci chez 10 malades En cas d'obstruction sous-entoriel (tumeur ou adherences dans la fosse cranienne posterieure) (Fig 3) chez 15 malades (sur un total de 28) le ommet de l'angle veineux etait leg erement deplace en bas tandis que chez les 13 autres malades ce ommet ■ trouvait a la place habituelle ans etre souleve chez aucun des malades L'angle veineux etait ensiblement ouvert chez 12 malades et leg erement ferme et en meme temps deforme en bec chez 5 malades On a constate chez 14 malades un certain aplatissement accompagne d'une

depression de la veine cerebrale interne une configuration et une disposition habituelles de celle-ci chez 13 malades et une arc anterieur accentuee chez un malade. On n'a constate cependant, chez aucun des malades de deformations en marche d'escalier ni un calibre irregulier ou une asymetrie. En outre chez 45 malades du 3eme groupe on a constate a divers degre une tension et un allongement de la veine du septum pellucidum et de ses affluents.

### Discussion

L'etablissement de criteres angiographiques en faveur du diagnostic d'hydrocephalie suscite automatiquement les deux questions suivantes (1) Quel est le caractere de l'hydrocephalie (communicante ou obstructive), et (2) s'il s'agit d'hydrocephalie obstructive quel est le niveau de l'obstruction (sur ou sous-tentorial).

Les resultats de notre recherche montrent que les alterations arterielles hydrocephaliques ont nettes surtout chez les malades manifestant une hydrocephalie nettement obstructive. Il est impossible cependant de juger du caractere de l'hydrocephalie et du niveau de l'obstruction uniquement sur la base des alterations arterielles. Tout de meme les modifications arterielles hydrocephaliques parlent plutot en faveur du caractere obstructif de l'hydrocephalie. D'autre part la presence d'une deviation discrete de l'artere pericalléuse par rapport a la ligne mediane montre qu'il existe un processus expansif dans le 3eme ventricule. Il nous semble que le signe propose par AGNOLI (1962) tension et deformation en arc accompagnees de convexite en avant du segment proximal inferieur de l'artere pericalléuse en cas de niveau sous-tentorial de l'obstruction represente un critere peu valable vu que ce signe a ete observe aussi chez 7 de nos malades manifestant un processus expansif dans la fosse cranienne posterieure (niveau sous-tentorial de l'obstruction) et inversement il n'a pas ete observe chez la moitie des malades souffrant d'hydrocephalie due a une tumeur dans le 3eme ventricule (niveau sur-tentorial de l'obstruction). Il ne faudrait pas compter non plus sur le soulèvement de l'artere cerebrale posterieure decrit par LOFGREN (1936) en cas d'hydrocephalie due a une tumeur du cervelet etant donne que lors de l'angiographie carotidienne d'apres les recherches classiques anciennes (15-20-21) on observe a peine chez 25-50% des cas une opacification de l'artere cerebrale posterieure.

Les alterations angiographiques hydrocephaliques du systeme veineux profond peuvent etre systematisees, sur la base des donnees de notre recherche en trois groupes.

1er groupe Alterations revetant un caractere pathognomonique — c'est la combinaison, exprimee par l'absence de deplacement de la veine cerebrale

interne par rapport à la ligne médiane et par la présence d'un déplacement latéral de la veine thalamo-striée, accompagnée d'une déformation en arc à convexité latérale de la même veine

2ème groupe Modifications présentant un caractère presque spécifique — nous y rapportons l'allongement et la tension de la veine du septum pellucidum et de ses affluents

3ème groupe Modifications à caractère non spécifique et variable — il s'agit de l'aplatissement ou de la configuration habituelle de la veine cérébrale interne, la dépression ou la disposition habituelle de cette même veine ainsi que des modifications de la valeur de l'angle veineux du cerveau et de la disposition de son sommet

C'est sur la base des alterations angiographiques hydrocephaliques du système veineux profond qu'on pourrait juger plus catégoriquement du caractère de l'hydrocephalie. Néanmoins la combinaison de modifications veineuses pathognomoniques (du phlebogramme de face), parlant en faveur d'une dilatation ventriculaire légère ou modérée avec l'absence d'alterations de la veine cérébrale interne et de l'angle veineux constatées sur le phlebogramme de profil montre qu'il s'agit d'une hydrocephalie communicante. Au contraire la combinaison de données du phlebogramme de face traduisant une dilatation ventriculaire massive avec celles du phlebogramme de profil montrant des modifications de veine cérébrale interne et de l'angle veineux témoigne que nous sommes en présence d'hydrocephalie obstructive

En ce qui concerne l'établissement angiographique du niveau de l'obstruction nous sommes d'avis qu'il pourrait être effectué avec une grande probabilité sur la base des modifications hydrocephaliques veineuses du 3ème groupe revêtant un caractère non spécifique et variable examinées cependant sur le fond des modifications hydrocephaliques veineuses pathognomoniques du 1er groupe. C'est ainsi que la combinaison de données provenant du phlebogramme de face et se rapportant à une dilatation ventriculaire massive avec celles du phlebogramme de profil concernant le soulèvement du sommet de l'angle veineux accompagnée d'une éventuelle ouverture ou fermeture de cet angle ainsi que d'un aplatissement éventuel avec soulèvement de l'arc antérieur de la veine cérébrale interne ou d'une déformation dans le trajet, le calibre ou les contours de cette veine tout cela parle en faveur du niveau sus-tentorial du niveau de l'obstruction (tumeur du 3ème ventricule). Au contraire la combinaison de données (du phlebogramme de face) traduisant une dilatation ventriculaire massive avec celles du phlebogramme de profil se rapportant à une dépression ou à une disposition normale (dans le sens vertical) du sommet de l'angle veineux accompagnées d'une ouverture éventuelle de cet angle de même que la présence d'un aplatissement éventuel avec dépression simultanée de l'arc



antérieur de la veine cérébrale interne, sans qu'il adviennent des déformations du trajet du calibre et des contours de cette veine traduisent la présence d'une obstruction sous tentorielle.

## RÉSUMÉ

Des recherches comparatives fondées sur les données des diverses phases et projections de l'angiographie carotidienne ont été effectuées chez 120 malade souffrant d'hydrocéphalie lesquels ont été soumis parallèlement à l'angiographie carotidienne et à la pneumographie. L'auteur a établi certains critères angiographiques sur la base desquels on peut déterminer avec une grande probabilité le caractère de l'hydrocéphalie (communicante ou obstructive) du même que le niveau de l'obstruction en cas d'hydrocéphalies obstructives (sus- ou sous tentorielle).

## SUMMARY

The pneumographic findings were compared with those obtained by carotid angiography in 120 cases of hydrocephalus. Certain angiographic appearances make it possible to conclude with some certainty the form of hydrocephalus that exists that is whether it is communicating or obstructive or in the latter instance whether it is located supra- or infra-tentorially.

## ZUSAMMENFASSUNG

An einem Material von 120 Fällen von Hydrocephalus wurden die pneumographischen Befunde mit den angiographischen Befunden verglichen. Es zeigte sich, dass bestimmte Zeichen bei der Angiographie es ermöglichen mit großer Wahrscheinlichkeit darauf zu schließen, welche Form von Hydrocephalus vorliegt, ob es sich um einen generellen Hydrocephalus handelt oder um einen Verschluss, und im letzteren Fall, wo der Verschluss lokalisiert ist, ob oberhalb oder unterhalb des Tentoriums.

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## VERTEBROBASILAR VENOUS SYSTEM IN THE ANGIOGRAPHIC DIAGNOSIS OF HYDROCEPHALUS

by

M. ROSA and G. L. VIALE

The venous phase of the carotid angiography, particularly as regards subependymal veins, may provide valuable evidence supplementary to the angiographic diagnosis in hydrocephalus (WOLF & HUANG 1964; HUANG *et coll.* 1968; ROSA 1968). Improvement in the knowledge of the roentgen anatomy of the vertebrobasilar venous system has led to further evaluations in the differential diagnosis of occlusive non tumoural hydrocephalus and communicating hydrocephalus. Although the venous phase in hydrocephalic infants does not substantially differ from that in adults, this paper is concerned only with the latter.

*Non tumoural hydrocephalus of obstructive nature.* Displacement of the veins of the vertebrobasilar system is mainly related to the subtentorial tumour that determines the triventricular hydrocephalus (HUANG *et coll.* 1965/68, VIALE & ROSA, ROSA & VIALE 1968/69/70, BRADAC *et coll.* 1969). Venous displacements of non tumoural obstructive hydrocephalus, on the other hand, are related to supratentorial ventricular enlargement. Deformation of the ventricles and increased pressure in the supratentorial space give rise to displacements of (1) subependymal veins which are tributaries of the internal cerebral vein, (2) the circummesencephalic venous system and Galenic vein, and (3) subtentorial



Fig 1 Controlled non tumoural hydrocephalus of obstructive nature (two different cases) Vertebral phlebogram lateral projection a) The circummesencephalic venous system particularly the mesencephalic posterior vein (←) is displaced downwards the precentral cerebellar vein (↔) is displaced backwards with inversion of its convexity b) The posterior mesencephalic vein (←) and the precentral cerebellar vein (↔) are displaced slightly downwards and backwards after surgical exploration of the fourth ventricle and Torkildsen drainage In this condition some compensation exists between the supra and subtentorial pressure

veins belonging to the anterosuperior surface of the cerebellar hemispheres and vermis These displacements are a consequence of the pressure in the region of the tentorial notch

The medial atrial vein is generally the only subependymal vein that is evident in vertebral phlebography It appears stretched and elongated in the lateral projection an aspect related to the dilatation of the trigone of the ventricle The circummesencephalic system particularly the mesencephalic vein and the Galenic vein is displaced downwards in the lateral projection (Fig 1) together with the veins of the anteroposterior surface of the cerebellum Those located on the lobulus quadrangulus the anterosuperior and marginal veins are moderately affected by the pressure while the more medial precentral vein presents marked deformation with inversion of its convexity (Fig 1 a) These factors represent complementary evidence in the differential diagnosis of communicating hydrocephalus, a condition in which some compensation exists between the supra and subtentorial pressure No further evidence is obtainable by a p views

*Communicating hydrocephalus* The most striking difference between phlebography in communicating and obstructive hydrocephalus is the absence of displacements of the circummesencephalic venous system and the veins of the anterosuperior surface of the cerebellum in the former (Fig 2 a) The veins of

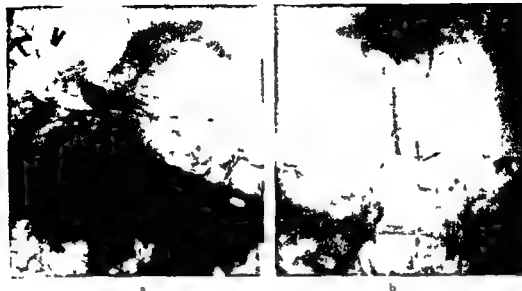


Fig. 2 Communicating post-traumatic hydrocephalus (previous temporo-parietal right brain laceration and intracerebral haematoma). Vertebral angiography following encephalography. a) Lateral projection. Absence of displacement of the circumferential venous system; the veins of the anterosuperior surface of the cerebellum and the precentral cerebellar vein. b) Ap projection. The vein of the left lateral recess of fourth ventricle ( $\leftarrow$ ) is displaced laterally; the supratentorial branch ( $\leftarrow$ ) is pulled upwards in relation to the enlargement of the fourth ventricle. The right vein is not seen.

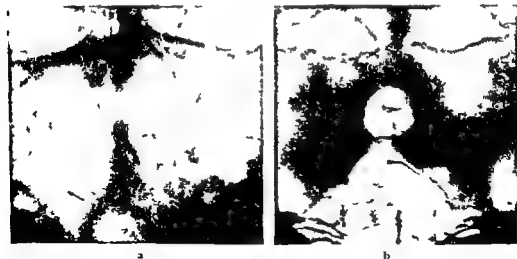


Fig. 3 Communicating hydrocephalus. a) Vertebral phlebography, lateral projection. The vein of the lateral recess of fourth ventricle ( $\leftarrow$ ) is deformed and displaced laterally in relation to the enlargement of the fourth ventricle (b).

the lateral recess of the fourth ventricle are deformed in relation to its enlargement (Figs 2 b and 3). Both veins are displaced laterally in the a.p. projection (Figs 2 b and 3 a). marked reduction in the ponto-cerebello-medullary angle thus occurs. The deformation of the veins is difficult to assess in the lateral projection but this view indicates that the anterior tributaries of the inferior vermian vein are dissociated following the dilatation of the fourth ventricle.

Communicating hydrocephalus in the adult produces appearances of the veins related to the fourth ventricle similar to those that result from a tumour of the fourth ventricle with occlusive hydrocephalus. Although the venous phase in this condition also assists in the differential diagnosis this is mainly dependent on other factors.

### Conclusion

The radiologic evaluation of the veins related to the brain stem, the fourth ventricle and the anterosuperior surface of the cerebellum, is of some value in non tumoural hydrocephalus. The differentiation between communicating and non tumoural obstructive hydrocephalus although founded on encephalography may be facilitated by use of the venous phase of vertebral angiography.

### SUMMARY

Evaluation of the veins of the vertebrobasilar system in relation to the mesencephalic structures, the anterosuperior surface of the cerebellum and the fourth ventricle contributes to the angiographic diagnosis of hydrocephalus. The signs obtained in the venous phase are particularly useful as regards the differential diagnosis between communicating and non tumoural obstructive hydrocephalus.

### ZUSAMMENFASSUNG

Die Darstellung der Venen des vertebrobasilaren Systems die in Beziehung zu den mesencephalen Strukturen der anterosuperioren Oberfläche des Kleinhirns und dem vierten Ventrikel stehen trägt zur angiographischen Diagnose des Hydrocephalus bei. Besonders wichtige Anhaltspunkte für die Differentialdiagnose zwischen kommunizierendem und nicht tumorbedingtem obstruktiven Hydrocephalus liefert das Phlebogramm.

### RÉSUMÉ

Étude des veines du système vertébro-basilaire qui sont en rapport avec les structures mésencéphaliques, la surface antéro-supérieure du cervelet et le quatrième ventricule pour le diagnostic angiographique de l'hydrocéphalie. Les signes donnés par le phlébogramme sont particulièrement utiles pour le diagnostic différentiel entre hydrocéphalie communicante et hydrocéphalie obstructive non tumorale.

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## COMPARISON OF TEMPORAL HORN WIDTH AND ISOTOPE CISTERNOGRAPHY IN THE EVALUATION OF VENTRICULAR ENLARGEMENT

by

O SJAASTAD, K. ROOTHELT and H. NORNES

The shape of the ventricular system has recently been demonstrated as being different in hydrocephalus ex vacuo and ordinary hydrocephalus due to pressure (SJAASTAD *et coll.* 1969). Ventricular enlargement in hydrocephalus ex vacuo usually seems to involve the upper part of the lateral ventricles leaving the temporal horns normal or only moderately enlarged. Generalized dilatation of the ventricular system including the temporal horns usually occurs in pressure hydrocephalus.

Two ratios may be used to indicate this difference in behaviour of the temporal horns in the two conditions: the third ventricle width/temporal horn width ratio and the ventricular body width/temporal horn width ratio. Since the temporal horns are relatively larger in pressure hydrocephalus than in hydrocephalus ex vacuo, the ratios will accordingly be lower in the former than in the latter. As for the third ventricular width/temporal horn width ratio, a ratio of 1.7 was found to differentiate best between the two types of ventricular enlargement: with this ratio the overlapping was only approximately 8 per cent.



Table

*RISA tests in cases of probable normal pressure hydrocephalus and hydrocephalus ex vacuo according to ratios*

	Total	Normal	Retention at site of injection	Retardation of flow	Ventricular re- flux and cortical flow	Ventricular re- flux only
Ratio indicating pressure in hydrocephalus	11			3	4	4
Ratio indicating hydro- cephalus ex vacuo	17	11	1	3	2	

Pressure is also believed to dilate the ventricles in so-called normal pressure hydrocephalus despite the fact that it is normal or only moderately elevated at the time fully developed hydrocephalus is demonstrated. If pressure is a decisive factor for the development of ventricular enlargement in this type of hydrocephalus the ratios should point to a pressure factor even in this condition.

A group of adults with marked dilatation of the ventricular system and normal intracranial pressure has been investigated. These were divided into those with possible hydrocephalus ex vacuo (a total of 17) and those with a possible pressure factor (11 patients in all) on the basis of the individual ratios. A RISA test was then carried out intrathecally in the lumbar region in each patient.

None of the 11 patients with a possible pressure factor according to the ratios had a definitely normal cerebrospinal fluid flow pattern with the RISA test; either retardation of flow was present or the ventricular reflux occurred alone or together with cortical flow (Table). Eleven of the 17 patients with possible atrophy had a normal flow and in none of these did ventricular reflux alone occur. Ventricular reflux together with cortical flow or retarded flow occurred in 5 patients. There is thus a highly significant difference in cerebrospinal fluid flow pattern between the two groups selected according to ratios ( $p < 0.001$ , chi-square test).

Several alternative explanations for the discrepancy between the RISA results and ratios in the possible hydrocephalus ex vacuo patients exist. One may be that the pressure pattern in the encephalogram had had insufficient time to develop; it may be a time-consuming process.

The ratios were borderline in 6 patients so that the classification on this basis

was dubious. Two of these patients had a normal flow pattern by the RISA test. 3 had cortical flow and ventricular reflux, whereas right sided Sylvian fissure block was present in one patient.

Considerable enlargement of the ventricular system must apparently have taken place in order to bring out either of the two patterns. A classification based on ratios can thus not be brought about in patients with only moderate ventricular enlargement. normal cerebrospinal fluid flow pattern was invariably present in 6 patients in this category.

On the premise that ventricular reflux is pathognomonic of normal pressure hydrocephalus, the data indicate that the dilatation of the ventricular system is often virtually due to pressure per se. There seems to be three possible hypothetical explanations for the apparent inconsistency between the findings of a pressure pattern and the presence of normal pressure: (1) increased intraventricular pressure has been present at some stage during the development of this type of hydrocephalus; (2) the tolerance of apparent normal pressure may be reduced in these patients; tolerance to intraventricular pressure would also appear in theory to follow a Gaussian distribution and a normal pressure in one patient may consequently be detrimental in another patient; (3) the water hammer effect of the pulsations from ectatic vessels encroaching upon the ventricular system may contribute in the development of normal pressure hydrocephalus (BREIG et coll 1967, EKBOM et coll 1969).

The ratios seem to be a useful parameter for evaluating even possible normal pressure hydrocephalus.

## SUMMARY

Twenty eight adults with ventricular dilatation and normal intraspinal pressure were grouped from the widths of the temporal horns into those with possible pressure hydrocephalus and hydrocephalus ex vacuo. A highly significant difference in the cerebrospinal fluid flow pattern existed between these two groups. This suggests that the dilatation of the ventricular system in so called normal pressure hydrocephalus is due to a pressure factor in spite of a recorded normal pressure.

## ZUSAMMENFASSUNG

Achtundzwanzig Erwachsene mit Dilatation der Ventrikel und normalem intraspinalen Druck wurden nach der Weite der Temporalhörner in solche mit möglichem Druck Hydrocephalus und solche mit Hydrocephalus ex vacuo aufgeteilt. Ein hochsignifikanter Unterschied im Verhalten des cerebrospinalen Flüssigkeitsstroms findet sich zwischen diesen beiden Gruppen. Das lässt vermuten, dass die Dilatation des Ventrikelsystems beim sogenannten Normaldruck Hydrocephalus durch einen Druck Faktor trotz des gefundenen normalen Drucks hervorgerufen wird.

## RÉSUMÉ

Les auteurs ont élisé vingt huit adultes atteints de dilatation ventriculaire avec pression rachidienne normale d'après la largeur des cornes temporales en malades pouvant avoir une hydrocéphalie avec pression augmentée et malades avec hydrocéphalie ex vacuo (par atrophie). Les auteurs ont trouvé entre ces deux groupes une différence très significative dans le type d'écoulement du liquide céphalo rachidien. Ceci fait penser que la dilatation du système est due à un facteur de pression malade. L'enregistrement d'une pression normale

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## DECREASE IN CEREBROSPINAL FLUID PRESSURE FOLLOWING THE PRESSURE INJECTION TECHNIQUE

by

K. SUGIURA K. OHNAMI Y. YABE and S. KONDO

The results of cerebrospinal fluid pressure measurements following two different types of encephalography have previously been reported (KONDO et coll 1970). First air was introduced intrathecally without removal of CSF throughout the procedure (pressure injection encephalography). The pressure fell soon after this procedure and almost always lasted for one to three days before returning to its original value. Secondly overflow encephalography was performed i.e. the CSF was allowed to flow out freely from a cut manometer tube to maintain the initial pressure when the air was injected into the lumbar sac. This group of patients had no appreciable pressure changes later.

Nitrous oxide or combinations of nitrous oxide and air were tried as contrast media by the pressure injection technique. Isotope investigations were also carried out to investigate the changes in the CSF dynamics that had occurred.

*Pressure injection of air* The technique of pressure injection encephalography is in itself not largely different from those advocated by LINDGREN (1949) or ROBERTSON (1965) in which gas is introduced fractionally without allowing the CSF to escape at any stage during the procedure. The patients were if necessary pretreated with atropine and placed in a sitting position. Lumbar

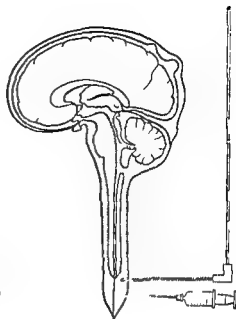


Fig. 1 Pressure injection encephalography. Two lumbar puncture needles and a manometer tube raised two meters high are depicted.

puncture was performed under local anesthesia at L4–L5. An additional needle was sometimes inserted at the level of L3–L4 for continuous recording of the pressure before and during the procedure either by observation through a long vinyl tube or by connection to a pressure transducer (Fig. 1). The return of the elevated pressure to its original value takes place immediately after the end of the gas injection even if no CSI is withdrawn (Fig. 2). This method of pneumography is called pressure injection because gas must be injected at a higher pressure to overcome the CSI pressure. The rate of air injection was 3 ml per minute and the total amount of air introduced ranged from 10 to 36 ml.

One and three days after the procedure lumbar puncture was performed in the lateral decubitus position in all the patients and the pressures thus obtained were compared to the originals. The CSI pressure decreased considerably one to three days after the procedure whatever the original value (Fig. 3).

The following cases from the pressure injection series may indicate why the authors suggest that this procedure is not only harmless but is sometimes beneficial to those who have evidence of high intracranial pressure or hydrocephalus.

*Case 1.* Male, aged 60 years with high intracranial pressure and papilloedema in poor condition and semicomatose. Lumbar puncture revealed a markedly elevated opening pressure of 220 mm of water. Pressure injection encephalography with 36 ml of air demonstrated

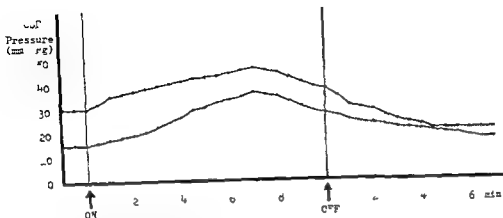


Fig 2 Pressure changes during pressure injection encephalography. Injection of nitrous oxide at a rate of 10 ml/min. The pressure falls spontaneously soon after the end of the gas introduction even if no CSF is removed.

a large bilateral frontal lobe meningioma. The CSF fluid pressure fell to 340 mm on the first day and 220 mm of water on the third day. Electroencephalography had previously disclosed diffuse slowing (Fig 4a) although the second EEG three days after the pressure injection indicated a return of some basic rhythm and the disappearance of gross bradycardia (Fig 4b). The patient became slowly but definitely conscious, could obey simple commands on the first day and converse after three days. Bifrontal craniotomy on the fourth day revealed a large bilateral meningioma with the dura for such a large tumour surprisingly soft.

**Case 2.** Girl aged 6 months with hydrocephalus for three months. The CSF measurements were not recorded but the head circumference and the diameters of the anterior fontanelle were measured every day before and after the pressure injection encephalography with 75 ml of air. The falls in the CSF pressure may have resulted from this procedure (Table 1).

**Overflow encephalography.** If a vinyl tube for manometric observation raised 2 meter above the lumbar puncture needle or connected to a pressure transducer is cut and opened at the level of opening pressure, any increased pressure on the CSF system during the procedure may be avoided. This method is termed overflow encephalography because the CSF flows out freely from the cut manometer tube when gas is injected into the lumbar sac. Eight consecutive patients with minor head injuries were examined. The amount of air introduced ranged from 36 to 100 ml (average 60 ml) which was considered enough to cause pressure reduction if applied by the pressure injection method. The lumbar CSF pressure on the other hand presented no appreciable changes after one to three days (Fig 5).



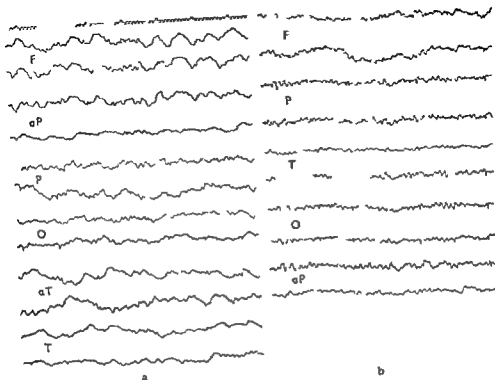


Fig 4 Case 1 a) Before and b) three days after pressure injection encephalography with 36 ml of air. Return of some basic frequency and reduction of gross bradycardia in (b)

procedure, almost the same as in pressure injection of air (Fig 6 b). The addition of 15 to 20 ml of air to nitrous oxide in the other patients also produced a relatively constant decrease even if the amount of the latter were small (Fig 6 c).

The conclusion to be drawn from these data is that the elevation of CSF pressure during the procedure is the most important cause of the decrease in pressure at 24 to 72 hours.

*Investigations of cerebrospinal fluid dynamics with RISA* RISA was introduced into the lumbar sac to determine the mode of CSF flow followed by the pressure injection. The movement of protein was observed by serial scanning originally described by Di Ciro et coll (1964) as PISA scintigraphy.

Twenty RISA injections into the lumbar sac of 10 patients have been investigated. The 10 patients first received 140  $\mu$ Ci of RISA diluted in 1 ml of saline in order to obviate any change in the CSF dynamics. About two weeks later 20 ml of saline were injected into the lumbar sac from one needle at the rate of



Table 1

Case 2. Head circumference and diameter of large fontanelle in hydrocephalus with evidence of falls in CSF pressure. Pressure injection encephalography was performed on the 7th day after a lumbosacral puncture

Day after lumbosacral	Head circum- ference (cm)	Diameter of large fontanelle (mm)	Body weight (kg)
1	44.0	63	6.000
2	44.5	67	
7			6.800
8	48.0	73	
10	49.0	74	
11	49.5	74	6.50
13	49.0	74	
15	49.0	75	6.50
17	49.0	76	7.00
18	49.0	76	6.50
21	49.0	75	7.00
22	49.0	75	7.20
23	49.0	75	7.370

1 ml per minute and the CSF pressure was continuously observed through another needle connected to a manometer tube. As soon as the pressure returned to its original level 1 ml of RSA containing saline was slowly installed into the lumbar subarachnoid space.

The results obtained are presented in Table 2 and in Fig. 7. It may be said that in general the cephalad flow of CSF exists irrespective of volumes of the saline injected, but the speed of its flow becomes much faster when 20 ml of saline are first injected, i.e. after pressure injection.

### Discussion

The fractional techniques used today were first described by LUNDHOLM (1919) and ROBERTSON (1965), although a number of techniques for performing lumbar encephalography had been advocated. However many physicians still prefer ventriculography to encephalography especially when a cerebral tumour is likely.

There is no doubt that the risk of herniation is one of the reasons why encephalography is not used widely in spite of its several advantages. Herniation will result when the intracranial pressure greatly exceeds the intraspinal pressure. This may occur as a result of an increase of the former by brain oedema caused by the irritative effect of air (DANBY 1957), or the removal of fluid

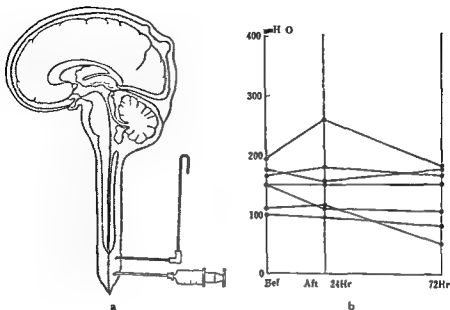


Fig 5 a) Overflow encephalography. A cut manometer tube lies at the level of the initial pressure b) Pressure changes before and after the procedure

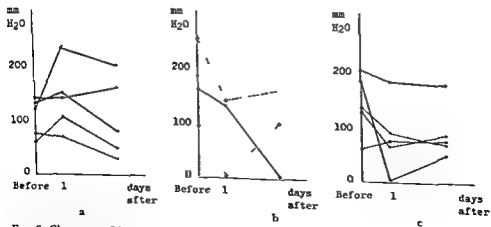


Fig 6 Changes in CSF pressure following the introduction of 40-50 ml of nitrous oxide (a) 90-120 ml of nitrous oxide (b) and 20 ml of nitrous oxide plus 15-20 ml of air (c)

Table 2

Areas of the head test accumulation of radioisotope. Numerals without parentheses indicate the time elapsed in hours after injection of KPS 1 diluted in 1 ml saline. Numerals in parentheses indicate hours after pressure injection of 20 ml saline in the same patient. The rapid flow of the tracer and its disappearance from the intracranial subarachnoid space in the latter are of parent

Age and sex	CSI pressure	Diagnosis	Areas of the head test accumulation of radioisotope				Time of disappearance
			Spinal subarach- noid space	Cisternal magnus	Basal cisterns	Superior sagittal sinus	
32 ♀	110	Mild head injury	1 h	3 (1 h)	6 (3)	24 (12)	(24)
17	110		1 h	3 (1 h)	6 (3)	24 (12)	(24)
23 ♀	110		1 h	3 (1 h)	6 (1 h)	24	(6)
21 ♀	110		1 h	3 (1 h)	6 (3)	24 (12)	(24)
33 ♀	110			3 h (1 h)	3 (1 h)	12	24 (6)
63 ♀	110		1 h	3 (1 h)	6 (3)	24 (12-24)	
27 ♀	110		1 h (1 h)	3 (3)	6	24 (Return of isotope into spinal subarachnoid space)	
33 ♀	110		1 h	3	6	24 (Rad. activity taken as end point of examination)	
49	200	Hydrocephalus narrowing shunt	RISA present and upward movement in subarachnoid space (RISA present and upward movement)				
3	120	Posttraumatic hydrocephalus	1 h	3-4 (1 h)	3 (3)	24 (12)	(24)

through a lumbar puncture needle as was formerly done when the block was not understood.

The maintenance of intraspinal pressure higher than intracranial pressure will thus become important in eliminating the hazards of herniation and will be accomplished theoretically by gas injection without the withdrawal of CSI. MESSERT et al. (1966) described the deterioration of hydrocephalic patients



Fig 7 Scans obtained in the same patient after the introduction of different amounts of saline into the lumbar subarachnoid space a) 3 hours and b) 6 hours after the injection of 1 ml of RISA containing saline c) 3 hours and d) 6 hours following the pressure injection of 20 ml The rate of flow of the radioisotope is slower and the concentration is less in (a) and (b)

after air encephalography or even after simple spinal tap ADAMS et coll (1965) also noticed in their hydrocephalic patients the deleterious effects of encephalographic examinations. Although these deteriorations were attributed to a sudden decrease in the pressure due to withdrawal of CSF during the procedure it should be remembered that if more than 30 ml of spinal fluid are rapidly removed its pressure following restoration to normal amounts to a higher level for from eight to twenty four hours (MASSERMAN 1934). On the other hand the injection of saline into the lumbar sac is known to be accompanied by a rise in pressure but is followed by a fairly rapid return to the preinjection level as soon as the injection is stopped even if no CSF is withdrawn (RYDER et coll 1953). It would appear however that no reports are available that the elevated pressure falls to less than the original level and continues to be low for some days, i.e. the reverse of MASSERMAN's findings.

The fall in CSF pressure after lumbar puncture may be due to leakage of fluid from the dural puncture holes (MACROBERT 1918). It should however be stressed that no appreciable pressure changes occurred at 24 and 72 hours in the overflow encephalographies notwithstanding the two lumbar puncture holes made in the spinal dura. The leakage of CSF from dural holes does not seem to play an important role in causing CSF hypotension following the pressure injection: the difference between this and the overflow lies not in the number or size of holes or in the way they are made but in the effect in the former of the undue hydrostatic pressure imposed on the CSF system during the procedure. The same explanation may be possible in the interpretation of the results of pressure measurements after the pressure injection of nitrous oxide into the lumbar sacs. Nitrous oxide due to its high water solubility may exert a pressure high enough to produce a later pressure alteration only when the volume injected exceeds its maximum solubility in the CSF and can exist in the fluid space as gas.

Since the time of its introduction by DI CHIRO *et coll* (1961) RISA cisternography has become an important aid in the field of neuroradiology. The radioactivity is detected in the basal cisterns 1 to 2 hours after injection, spreads over the hemisphere at 6 hours, accumulates along the superior sagittal sinus at 12 to 24 hours, and disappears at 48 hours. DI CHIRO, in his original report, stressed that the amount of solution injected constitutes the most important factor in its cranial passage after an intrathecal injection and recommended the dilution of the RISA of up to 10 to 20 ml or more. ISHIBASHI (1959) reported that no proof existed of the existence of cephalic CSI flow after the intrathecal injection of small amounts of RISA containing fluid. CHOU & LARSON (1955) stated however that a cephalic diffusion occurs along the neural axis after an injection of RISA in volumes of 2 ml although the slow process takes at least 20 to 24 hours to reach equilibrium in all the CSI compartments.

The present isotope investigations have indicated that RISA usually passes upward from the lumbar subarachnoid space whether the injected volumes are large or small. The rate of CSI flow into the cranial subarachnoid space and its disappearance from there however becomes considerably slower after the introduction of RISA in a volume of 1 ml compared with that after the pressure injection of 20 ml of saline. It is evident from the data that the volume of injection is important because it changes the speed of the CSI flow. It would appear that the normal flow pattern previously described by many authors following the injection of similar large volumes, is not physiologic in a strict sense but the result only of altered CSI dynamics. The mechanism whereby a faster flow is brought about after pressure injection may be explained from dynamics altered by the forced elevation of the CSI pressure gradient with a resulting increased fluid drainage into the blood stream.

MASSERMAN (1934) and RADER *et coll* (1953) considered that the return of an elevated CSI pressure to a previous level is due to changes in volumes of the craniospinal vascular bed. TORRES & ARROWOOD (1948) reported that a steady state is reached with the pressure higher than the original value if fluid is injected continuously, under the conditions the raised pressure is sufficient to cause an increased drainage equal to the amount of fluid injected. The present authors also believe that all of these explanations may hold true during or immediately after a forced injection of gas or fluid. However after the pressure returns to its original level it is difficult to look upon the above mechanisms as being responsible for the further pressure lowering. These considerations have suggested that increased CSI drainage should be the result of decrease of resistance at the site of absorption and brought about by high pressure exerted on the subarachnoid spaces.

DANDY & BLACKMAN (1917) stated communicating hydrocephalus is caused

by a barrier of adhesion at the base of the brain which mechanically prevents the CSF from reaching the site where absorption takes place BAGLEY (1928) from clinical and experimental investigations considered a block in the subarachnoid space through which the CSF returns to the blood stream to be the cause of hydrocephalus after subarachnoid hemorrhage. The possible role of obstruction of the arachnoid villi in the pathogenesis of occult hydrocephalus was also suggested (ELLINGTON & MARGOLIS 1969). It is reasonable to assume that pressure injection may cause the destruction of microstructures of arachnoid villi as was observed by WELCH & FRIEDMAN (1960) and reopen the obliteration of the subarachnoid space either over the cerebral convexities or at the basal cisterns or establish alternate routes that are capable of absorbing CSF. In the pressure injection series only exceptionally was no change in pressure present after the procedure. One of the patients had suffered from multiple subarachnoid hemorrhages; this may have caused a block of the subarachnoid space or arachnoid villi so severe that the additional pressure in the lumbar region could not produce the mechanism described.

JIROUT (1956) reported that the lateral recess of the lateral ventricle was considerably narrowed in the last film obtained during encephalography as compared with the first film taken immediately after the air was introduced. This author thought that this was the result of edema of the ventricle walls caused by the irritative effect of air. The present authors would express no opinion upon this concept although the investigations indicate that the effect of an increase in CSF drainage exceeds that of edema at least for several days after the pressure injection. This is disclosed not only by the sustained pressure lowering but also by the electroencephalographic and clinical signs of improvement in most patients with high CSF pressure before the procedure.

### Conclusions

A fall in CSF pressure below the initial level takes place after the pressure injection of air or fluid. The investigation suggested that no CSF leakage occurred through the lumbar puncture holes and stressed the role of increased fluid drainage from the craniospinal space; this was due to a decrease in resistance at the site of fluid absorption brought about by an unusual hydrostatic pressure imposed on the CSF system. An elevation of pressure during the pressure injection of small amounts of nitrous oxide, is not sufficient to cause a decrease of resistance in the fluid drainage due to the high water solubility of the gas. This correlates well with the results of overflow encephalography in which theoretically no pressure is exerted during the procedure.

It was ascertained by using RISA as a tracer that a faster CSF flow results from the pressure injection of saline. Its mechanism was explained by the altered dynamics produced by the forced increase in the pressure gradient with a resulting increased fluid drainage into the blood stream.

The authors consider that pressure injection cephalography is not harmful to patients who have high intracranial pressure because of its depressive effect on the intracranial pressure for several days. Further observations in hydrocephalus following head injury or subarachnoid hemorrhage have suggested that this pressure injection may even favourably alter the clinical course of developing hydrocephalus.

## SUMMARY

It is established that a fall in CSF pressure below the initial level takes place one to three days following the intrathecal injection of air without the withdrawal of fluid (pressure injection). The authors attribute this fall of increased fluid drainage from the subarachnoid space due to a decrease in resistance to the flow of fluid absorption. This appears to be brought about by an unusually high hydrostatic pressure exerted on the CSF system.

## ZUSAMMENFASSUNG

Es ist nachgewiesen, dass ein bis drei Tage nach einer intrathekalen Injektion von Luft ohne Entnahme von Flüssigkeit (Druck-Injektion) der CSF-Druck unter das initiale Niveau sinkt. Die Autoren behaupten, die Bedingung, gesteigerter Flüssigkeitsdrainage vom subarachnoidalen Raum als Folge des verminderten Widerstandes an der Stelle der Flüssigkeitsabsorption. Dies scheint durch einen ungewöhnlich hohen hydrostatischen Druck auf das CSF-System ausgeübt wird hervorgerufen zu werden.

## RÉSUMÉ

Il est établi que la pression du liquide céphalo-rachidien baisse au-dessous du niveau initial entre un à trois jours après l'injection dans les espaces meninges d'air sans succion de liquide (injection sous pression). Les auteurs soulignent le rôle de l'augmentation de la résistance du liquide céphalo-rachidien due à une diminution de la résistance à l'écoulement des fluides. Ceci paraît dû au fait qu'une pression hydrostatique inhabituellement élevée exerce sur le système du liquide céphalo-rachidien.

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## EXPLORATION TOMOÉCHOGRAPHIQUE INSTANTANÉE DE L'HYDROCEPHALIE DU NOURRISSON

par

F. WEILL, J. BECKER, J. F. BONNEVILLE et R. STEINLE

Un certain nombre de très belles images tomoéchographiques cranio-encéphaliques ont été publiées ces dernières années (ADAROV et coll 1965, GROSSMAN 1966, MAMO et coll 1968). Pourtant la tomoéchographie n'est pas encore devenue une technique neuroradiologique courante.

Les ultrasons se réfléchissent dans les parties molles. De ce fait les processus tumoraux sont en principe directement accessibles à l'exploration. L'importante réflexion du faisceau sur la paroi crânienne, les pertes d'énergie survenant dès que le faisceau n'est plus normal à la structure examinée, la survenue d'échos diffusés rendent difficile l'obtention d'images sûres et constantes dans ce domaine.

Une moins grande ambition est mieux récompensée : il est possible chez l'enfant d'obtenir des images sûres et constantes des parois ventriculaires. Celles-ci apparaissent comme des éléments linéaires symétriques par rapport à la ligne médiane (Fig. 1). Des coupes horizontales successives font apparaître le troisième ventricule et différents segments des ventricules latéraux. Notre appareillage est tomoéchographique. L'image est continue et apparaît de façon immédiate avec ses différents éléments pour chaque position du palpeur ultrasonore (Vidoson, Siemens). De ce fait l'exploration est rapide : dès que l'orientation normale de la sonde est trouvée, les images des différents plans sont construites en quelques minutes.

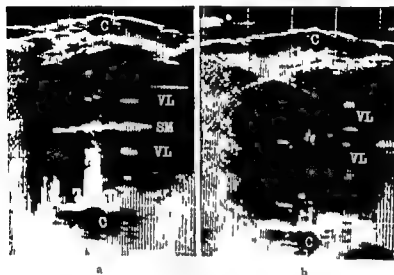


Fig 1 Coupes horizontales du crane chez un enfant de 8 jours a) Cornes frontales des ventricules lateraux b) Coupe passant par les carrefours ventriculaires C — paroi cranienne VL — paroi des ventricules SM — structure mediane

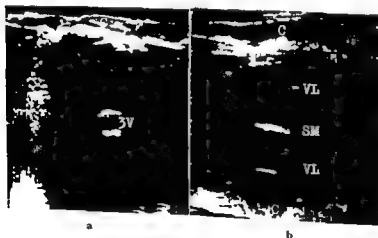


Fig 2 Coupes horizontales du crane chez une enfant de 5 ans presentant une hydrocephalie secondaire à un cranio-pharyngiome a) Coupe echotomographique passant par le troisieme ventricule b) Coupe passant par les ventricules lateraux dilates C — paroi cranienne 3V — troisieme ventricule VL — paroi du ventricule lateral SM — structure mediane

L'appareillage a par contre un défaut: il ne permet pas d'examen satisfaisant après 15 mois, du moins lorsque les structures encéphaliques sont normales. En cas d'hydrocéphalie, la diminution de l'épaisseur de tissu cérébral a traverser autorise des succès bien plus tard, et de ce fait nous avons dans ces conditions pu recueillir des documents excellents jusqu'à l'âge de 5 ans (Fig. 2).

La mesure ventriculaire est immédiate grâce à l'étalonnage direct du l'écran tomoecho copique. Les confrontations pneumographiques montrent que la méthode est suffisamment fiable pour rendre inutile l'examen contraste dans la simple appréciation des dimensions ventriculaires, en particulier dans la période de surveillance post opératoire après dérivation.

## RÉSUMÉ

L'utilisation d'un appareil tomoecho copique permet une mise en évidence immédiate des limites des ventricules latéraux chez l'enfant. Ceci permet le diagnostic et surtout la surveillance des hydrocéphalies notamment après dérivation et rend ainsi inutile un certain nombre d'études pneumographiques.

## SUMMARY

A tomoechoscope has been employed to indicate directly the margins of the lateral ventricles in children. This enables the diagnosis as well as the control of hydrocephalus particularly after tapping. It also enables a certain number of encephalographies to be avoided.

## ZUSAMMENFASSUNG

Ein Tomo-Echoskop wurde verwendet um direkt die Grenzen der lateralen Ventrikel bei Kindern festzustellen. Dieses Verfahren ermöglicht die Diagnose wie auch die Kontrolle des Hydrocephalus besonders nach Flusskeitsabnahme. Sie erubrigt ausserdem in einer Anzahl von Fällen ein Encephalographie.

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## CONTROL OF HYDROCEPHALUS BY INTRAVENTRICULAR RADIOACTIVE COLLOID

by

M H WEISS F E NULSEN and B KAUFMAN

The description in 1952 of valve regulated diversion of CSF has enabled the evolution of a program of successful surgical management of hydrocephalus (NULSEN & SPITZ 1957). More recent follow up investigations of patients so treated reveal that they may lead productive lives but only when continued control of ventricular size and pressure has been adequate, emphasizing the need for careful control of hydrocephalus. This surgical management of the problem is fraught with several problems the most frequent of which are shunt dysfunction related to growth and occasional infection with the internalized prosthesis. In an effort to obviate these complications a number of shunt revisions are necessary in the long term surgical management. This has led those interested in the problem to continue to look for alternative modes of treatment.

The choroid plexus is generally accepted as the source of production of a significant proportion of CSF (BERING 1958), and consequently numerous efforts have been directed at the ablation of choroid plexus in an effort to alter CSF dynamics. There have been reports of surgical ablation of choroid plexus both ex

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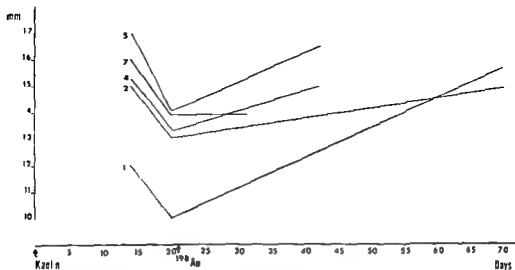


Fig 3 Cortical mantle Initial points represent cortical mantle at the time of the first ventriculogram. The graph then shows the consistent decrease in cortical mantle with time and then a return towards a normal cortical mantle after the instillation of  $^{199}\text{Au}$

determined program at 5, 10, 21, and 49 days after infusion of the colloids, the brain removed, fixed in formalin, and examined for histopathologic change.

The gamma energy component of  $^{199}\text{Au}$  enabled further investigation by gamma scanning of an additional 4 hydrocephalic animals as well as 2 children with severe degrees of hydrocephalus who received tracer amounts of radioactive gold. In addition, in these four animals, matched weighed specimens from multiple areas were assayed in a Packard liquid scintillation counter to evaluate radiation activity levels at specific anatomic sites. The absolute counts were converted to counts per gram tissue to provide a scale of relative activity.

## Results

Three of the animals in the first group did not survive the kaolin injection, leaving 7 animals for investigation.

**CSF pressures (Fig 2)** The mean CSF pressure as measured at the cisterna magna was 116 mm with a range of 90 to 135 mm. Following development of hydrocephalus before treatment with radioactive gold, the CSF pressure rose markedly with a mean of 175 mm and a range of 130 to 250 mm as measured at the lateral ventricle. Following instillation of the radioactive colloidal gold the intraventricular pressure fell dramatically in all cases but one with a mean of 76 mm and a range of 25 to 170 mm. It should be noted that values in all ani-

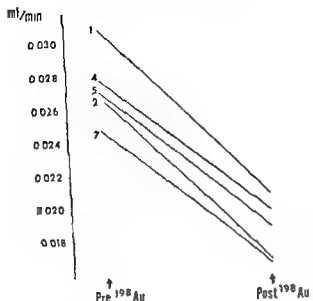


Fig 4 CSF flow Initial flow averaged 0.028 ml/min from the ventricles. Following treatment with  $^{198}\text{Au}$  flow fell by a mean of 32% to a mean rate of 0.019 ml/min.

imals except Nos 3 and 6 were obtained at 10 days after radioactive gold instillation and that, if the values for the 2 animals measured at 3 days are discounted, the mean is 39 mm with a range of 25 to 60 mm.

**Cortical mantle (Fig 3)** In all 5 animals followed beyond 5 days post treatment serial ventriculography showed progressive ventricular enlargement with consequent decrease in cortical mantle. Four of the five animals showed a subsequent decrease in ventricular size beginning at some time between 10 and 21 days after gold instillation. The one animal who was sacrificed early at 10 days post instillation showed an apparent arrest in the progressive ventricular enlargement although one could not demonstrate decreasing ventricular size at this time.

**CSF flow (Fig 4)** Average rate of CSF flow from the ventricle prior to treatment with radioactive gold was 0.028 ml/min with a range of 0.025 to 0.031 ml/min. Following treatment with radioactive colloidal gold (10 days) flow rates fell to an average of 0.019 ml/min with a range of 0.017 to 0.021 ml/min. This represented an average fall of 31.8% post treatment with radioactive colloid.

**Histologic investigation** of the removed brains revealed marked necrosis of choroid plexus with preservation of ependyma but marked vascular fibrosis. There was however particulate gold found in subependymal perivascular areas.

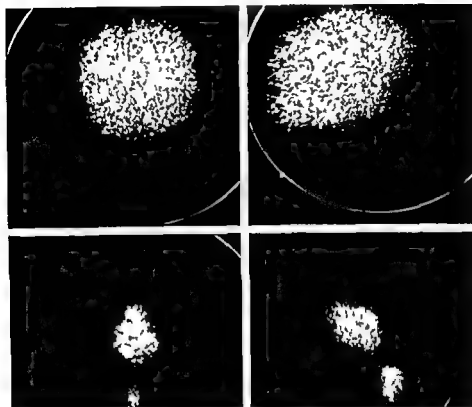


Fig. 3. Camera scan at 18 hours after intraventricular instillation of  $^{199}\text{Au}$ . Upper scans show supra and right lateral views of a hydrocephalic child and lower scans show subment supra and left lateral views in the same.

throughout the ventricular system although spectral scanning showed no evidence of subependymal cellular disturbance or demyelination up to 7 weeks after instillation. A more detailed description of the histopathologic changes forms the basis of another report.

Although the marked pathologic changes were observed exclusively in the choroid plexus the gamma scan which was performed daily for 5 days following instillation of the  $^{199}\text{Au}$  failed to reveal significant selective uptake in the area of the choroid plexus in either the animals or patients (Fig. 5). Sampling of tiny quantities of CSF at 24 hours post instillation revealed only about 10% residual activity in the CSF whereas one would expect greater than 50% activity remaining at this time interval if the colloid had remained in suspension within the ventricles.

Our expectation that the colloid deposits diffusely is confirmed by scintillation

investigations The per unit weight activity in the lining of the ventricles is almost half as great as the choroid plexus although tissues at depths of 5 to 10 mm from these lining surfaces contain less than 1 % of the plexus activity The relative values of the  $^{199}\text{Au}$  localization in various areas of the brain were choroid plexus 100 % roof of 4th ventricle 43 % lateral ventricular wall 38 % 3rd ventricular wall 35 %, aqueduct 34 %, floor of 4th ventricle 29 % caudate wall 28 %, cerebellar surface 4 %, frontal cortex and corona radiata 1 % each, mid thalamus cerebral peduncle, mid pons and cerebellum 0 % each

### Discussion

In the search for alternative techniques in the treatment of hydrocephalus the concept of selective radionecrosis of choroid plexus is quite appealing BERING has shown experimentally that surgical resection of the choroid plexus will reduce CSF production by about 28 % Our finding of an approximate 32 % reduction in CSF flow following choroid plexus radionecrosis is certainly in accord with those of BERING particularly when one considers the fact that the 4th ventricle plexi were ablated in our experimental model The reduction in CSF flow is also reflected in the sustained fall of intraventricular pressure along with a reversal of progressive ventricular enlargement

These results so far appear to be encouraging in considering  $^{199}\text{Au}$  as a possible agent in the clinical control of selected cases of hydrocephalus However the gamma scan findings are somewhat disturbing in certain respects The isotope failed to show selective concentration in the choroid plexus in follow up of 5 days after instillation well beyond its half life of 65.8 hours The finding of less than 10 % activity in remaining CSF at 24 hours however, indicates that the isotope is not remaining in suspension within the ventricles, but is apparently depositing diffusely within the subependymal tissue surrounding the ventricles Evidence of this can be found on histopathologic examination of the treated brains and is established even more precisely by the scintillation investigations which define a relatively generalized level of radioactivity throughout the ventricular lining

Our histopathologic findings would seem to incriminate vascular necrosis as the essential feature of choroid plexus ablation in the chronic animal (7 weeks after instillation) We were unable to demonstrate any evidence of vascular damage in areas other than the choroid plexus, even though one could demonstrate particulate gold in perivascular spaces Inspection of these areas reveals the particulate matter to appear less concentrated than in the areas of choroid plexus, and consequently disparate energy dosage may well account for the limitation of vascular fibrosis to the choroid plexus However, HAYMAKER (1968) has demonstrated delayed radionecrosis of cerebral blood vessels as long as 17 weeks



after radiation from external radiation source, and it is therefore conceivable that trophic changes might still occur in other tissues due to vascular compromise occurring even later than the 7 weeks for which our longest survivors were followed.

In addition to concern regarding the diffuse intraventricular deposition of radioactive material with the possibility that subtle subependymal pathologic and physiologic changes could ultimately result in the human, further management problems are raised by the amount of penetrating gamma radiation which accompanies an amount of radioactive gold sufficient to provide therapeutically effective beta radiation. If hospital personnel and parents must be protected from radiation while ministering to the infant for several days after injection it is difficult to escape the implication that risks to the patient's brain are being ignored. Safe extrapolation of this principle of choroid radionecrosis to humans would require (1) the finding of an equally ideal beta emitter with far less associated gamma activity and (2) the finding of a colloidal preparation whose physical properties favored more selective localization in choroid, or proof in far longer term experiments carried into primates that fortuitous deposition of radioactive material throughout the ventricular ependyma did not cause significant late radionecrosis in subependymal tissue.

This investigation validates the concept that radionecrosis of the choroid plexus is both technically feasible and physiologically effective in significantly reducing the production of CSF and in turn aiding in the control of obstructive hydrocephalus. It remains to define further the precise parameters involved and to assess fully the potential long term effect on other anatomical structures.

### SUMMARY

Hydrocephalic dogs treated with radioactive colloidal gold have been shown to have a sustained decrease in CSF flow and intraventricular pressure along with a reversal of progressive hydrocephalus. Gamma scanning and isotopic sampling however indicate a more diffuse distribution of the isotope than previously thought although pathologic changes up to 7 weeks post installation of the radioactive colloid appear confined to the choroid plexus.

### ZUSAMMENFASSUNG

Hunde mit Hydrocephalus die mit radioaktivem colloidalen Gold behandelt worden waren zeigten ein anhaltendes Absinken des Zuflusses der cerebro-spinalen Flüssigkeit und des intraventriculären Druckes verbunden mit einer Umkehr des progressiven Hydrocephalus. Gamma Scanning und Isotop-Proben deuten jedoch auf eine mehr diffuse Verteilung des Isotops als bisher angenommen obwohl pathologische Veränderungen bis zu 7 Wochen nach Verabfolgung des radioaktiven Colloids auf den Plexus chorioideus begrenzt zu sein scheinen.

## RÉSUMÉ

Des chiens hydrocéphales traités par l'or radio actif colloïdal ont présenté une diminution durable de la production de liquide céphalo-rachidien et de la pression intra ventriculaire avec une régression de l'hydrocéphalie. La scintigraphie et l'étude isotopique cependant montrent que la distribution de l'isotope est plus diffuse qu'on ne pensait bien que les modifications pathologiques jusqu'à 7 semaines après l'installation du colloïde radio-actif paraissent limitées au plexus choroïde.

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## RADIOACTIVE ISOTOPES



## DIFFERENTIAL DIAGNOSIS OF AN ABNORMAL BRAIN SCAN

by

G J ALKER JR, E V LESLIE and L BAKAY

The abnormal accumulation of radionuclide on a brain scan is non specific merely indicating the presence of abnormal tissue. Because of this brain scans are often reported as showing a lesion in a particular portion of the brain without an attempt made to diagnose the nature of the disease process. We feel that the experienced neuroradiologist, given a single set of abnormal brain scans can determine the nature of the lesion in most instances the accuracy being in the order of that of other neuroradiologic procedures such as angiography and pneumography.

*Materials and Methods* This conclusion is based on the review of over 3 500 brain scans performed in the Radiology Department of the Meyer Memorial Hospital over the past five years. Most of the scans were performed using  $^{203}\text{Hg}$  or  $^{199}\text{Au}$  tagged chlormerodrin as the pharmaceutical and the rectilinear scanner as the scanning instrument. We occasionally use technetium and the Anger camera.



Fig 1 Left frontal glioblastoma



Fig 2 Right parietal infarct involving a branch of the right middle cerebral artery

### Discussion

The interpretation of a single brain scan depends on the size, shape, location and density of the abnormal uptake as well as on the sharpness, number of lesions and the time relationship of the onset of neurologic signs and symptoms to the time of the examination. The availability of isotope flow examinations and serial scans over weeks or months add further valuable information to the diagnosis, but this paper is concerned with the evaluation of a single positive scan as performed in most hospitals with methods generally available.

Pertinent facts of the patient's history and neurologic examination should be available to the physician interpreting the scan and such information should be

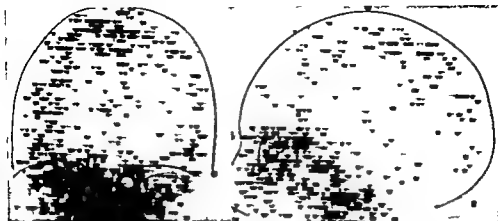


Fig 3 Right sided chronic subdural hematoma

supplied with the request for the examination. A thorough knowledge of neuroanatomy and pathology are essential.

*Size* Because of the inherent limitation of resolution of the scanning instruments lesions less than 1.5 to 2 cm are impossible to detect. Most lesions in our series were over 3 cm in diameter. Once recognizable, however, the size of the uptake does not affect the differential diagnosis.

*Shape* The lesion may be spherical, ovoid, crescentic, band like or wedge shaped and its shape in at least two planes must be considered. Neoplasms are almost always spherical or ovoid (Fig 1) whereas most non neoplastic lesions are not. Infarcts are classically but rarely wedge shaped in the lateral view and usually flat or crescentic in the anterior or posterior projections (Fig 2). Notable exceptions are infarcts in the occipital poles and in the region of the internal capsule which tend to be spherical and therefore indistinguishable from tumors. Abscesses are also spherical but much less common than tumors. Intracerebral hematomas though mostly spherical are seldom demonstrable at all on brain scans in our experience. Subdural hematomas the best known of the so-called crescent uptakes in the anterior view, usually reveal no localized lesion on the lateral (Fig 3).

Several convexity arteriovenous malformations were successfully diagnosed on the basis of brain scanning by noting that although there is a rounded lesion in the lateral view there is a rounded lesion plus a crescent in the anterior projection (Fig 4). The crescent represents the increased blood pool in the large supplying arteries and draining veins. Given a history of long standing seizure disorder and no significant shift of the midline structures the diagnosis is virtually certain.





Fig 4 Left parietal arterio venous malformation supplied by the middle cerebral artery

**Location** This is an important factor to consider in the differential diagnosis. The vast majority of infarcts occur in the territory supplied by branches of the middle cerebral arteries. Areas where the anterior and middle and posterior and middle cerebral artery branches overlap are unlikely to be affected because of collateral blood supply. A frontal or parietal parasagittal lesion, for example, is not likely to be an infarct. Meningioma, however, is much more likely at those sites. In order to diagnose an infarct, the lesion must not cover the territory supplied by more than one major vessel and must not extend deeply to an area usually occupied by a ventricle. As mentioned before, in the occipital poles and in the area of internal capsule, differentiation of tumor from infarct or intracerebral hematoma is virtually impossible. Infarcts in the posterior fossa, in the brain stem and in the area supplied by the anterior cerebral arteries are rarely a consideration in the differential diagnosis, and in fact these have seldom been demonstrated.

A posterior fossa lesion is almost certainly neoplastic. Subfrontal lesions are usually meningiomas although subfrontal extension of pituitary tumor may be considered. Lesions in the base of the skull may be a sphenoid wing meningioma as against a bone tumor, either primary or metastatic. Bone scan is useful for the differentiation. An osteochondroma, for example, has relatively low uptake on the  $^{203}\text{Hg}$  scan (Fig 5a), but is well demonstrated on the  $^{87}\text{Sr}$  bone scan (Fig 5b). Meningiomas tend to exhibit affinity to mercury as well as to strontium.

A lesion crossing the midline is not an infarct (Fig 6). About a third of glioblastomas involve the corpus callosum. Bull described this as causing a butterfly distribution of the radionuclide on the anterior scan. Falx meningiomas extend to either side of the midline as may deep seated frontal tumors resulting in a midline shift, but these are rounded in shape.

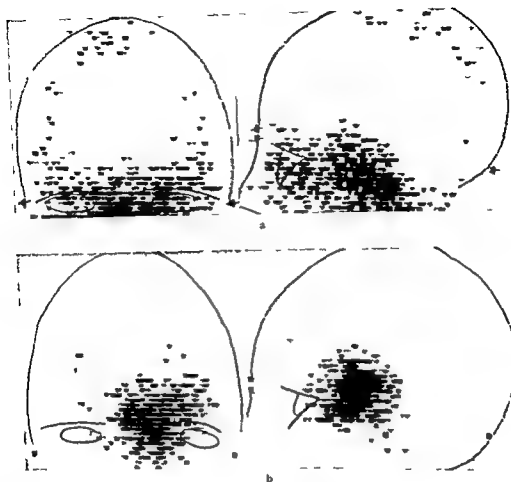


Fig 5 Osteochondroma of the base of the skull scanned with  $^{203}\text{Hg}$  (a) and  $^{86}\text{Sr}$  (b)

**Uptake** This factor must be evaluated cautiously since technical factors in instrumentation affect it. The relative display of normal and abnormal accumulations of the isotope on a given scan is subject to great variation due to individual preference of the examiner. With this in mind, infarcts exhibit a wide variation in uptake. Generally, the larger the lesion the denser the uptake. Glioblastomas almost always have an intense collection, whereas meningiomas are less uniform in this respect. Metastases have moderately intense uptakes. Low grade astrocytomas, craniopharyngiomas and pituitary tumors have little if any accumulation of isotopes.



Fig. 6 Left parietal glioblastoma infiltrating to the right by way of the corpus callosum

*Sharpness of margins* This is another variable quality influenced by technical factors, such as the speed of the scanning probe. In general, most tumors have a sharply outlined border except those that are diffusely infiltrating. The margins of small infarcts tend to be indistinct but these are on the surface in contrast to infiltrating tumors which extend deeply.

*Number of lesions* Multiple lesions are almost always metastatic tumors although an occasional instance of multiple infarcts due to emboli and multiple abscesses may be encountered. Of course a subdural hematoma with contralateral contusion is a double lesion but the shape and location of the uptakes help in the differential diagnosis. Rare cases of multiple meningiomas or tumors co-existing with vascular lesions may cause errors of interpretation.

*The factor of time* The timing of the scan in relation to the onset of neurologic signs and symptoms is very important in the differential diagnosis. For this reason it is important that this information be supplied with the request for the scan. It is well known that infarcts and subdural hematomas are not demonstrable on a brain scan for the first several days but are shown in most instances if the examination is performed at the optimum time, sometime during the second or third week of illness. Eventually, in the case of infarcts, the scan reverts to normal within a few weeks or months. It remains persistently positive in subdural hematoma for some time longer, even postoperatively. As to tumors, if untreated, the scan remains positive indefinitely. A positive uptake a day or two after onset of illness is almost certainly not an infarct.

## SUMMARY

The authors present the factors considered by them in the interpretation of a positive brain scan. No diagnostic method has an accuracy of 100%. By careful analysis of the brain scan, considering the factors outlined, the experienced neuroradiologist can expect to be correct in about 90% of his interpretations. This is on the same order of accuracy as with other neuroradiologic procedures such as angiography and pneumography. Combining the information obtained from brain scanning with that obtained from other procedures greatly enhances the accuracy of neuroradiologic diagnosis.

## ZUSAMMENFASSUNG

Die Faktoren, die von den Autoren für die Deutung eines positiven Gehirns Scans als wesentlich angesehen wurden, werden präsentiert. Keine Methode ist 100%ig genau. Bei sorgfältiger Analyse eines Gehirns Scans ist wenn die festgelegten Faktoren berücksichtigt werden, damit zu rechnen, dass der erfahrene Neuroradiologe zu etwa 90% in seinen Deutungen richtig ist. Das liegt in etwa derselben Größenordnung der Genauigkeit wie sie mit anderen neuro radiologischen Methoden wie Angiographie und Pneumographie erhalten wird. Kombiniert man die durch Gehirnschanning gesammelten Informationen mit denen von anderen Verfahren, lässt sich die Genauigkeit der neuroradiologischen Diagnose wesentlich verbessern.

## RÉSUMÉ

Les auteurs présentent les facteurs qu'ils considèrent comme importants dans l'interprétation d'une scintigraphie cérébrale. Aucune méthode est exacte à 100%. L'étude soignée de la scintigraphie cérébrale en tenant compte des facteurs choisis par les auteurs permet aux neuroradiologues expérimentés d'espérer faire un diagnostic correct dans environ 90% des cas. Ceci est du même ordre d'exactitude que les autres méthodes neuroradiologiques telles que l'angiographie et la pneumographie. L'association des informations fournies par la scintigraphie cérébrale aux informations données par les autres techniques augmentent de façon importante l'exactitude du diagnostic neuroradiologique.

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## BRAIN SCANNING AND CONVENTIONAL NEURORADIOLOGIC METHODS IN INTRACRANIAL TUMOURS

Scanning with  $^{99}\text{Tc}^m$  and a gamma camera

by

M BOWALLIUS, A LARSSON, H SKULDBORN and I WICKBOM

The diagnostic accuracy of the conventional neuroradiologic investigation and that of brain scanning have often been compared. The figures have varied considerably which may partly be explained by different techniques and sometimes by variation in the experience of the investigator. The accuracy of conventional methods appears to be lower, sometimes much lower (McAFFEE & FANDAL 1961, WANG 1969, YAMASAKI et coll. 1969) than in the present material from the period 1 March 1968 to 31 December 1969 performed with fairly uniform techniques.

**Methods** The brain scanning is performed after the patient has been premedicated with 400 mg potassium perchlorate and 0.5 mg atropine half an hour before an intravenous injection of 10 mCi  $\text{Na } ^{99}\text{Tc}^m\text{O}_4$ . The recording is started 45 to 60 minutes following the injection with a Pho gamma III equipped with PM tubes with a S 11 response. Registration is usually made in five projections: lateral (one from each side), p.p. and one from above (vertex), in

Table 1

Results of brain scanning angiography and encephalography in 188 patients with supratentorial tumours  
 Figures in parentheses indicate patients with multiple metastases

	Brain scanning			Angiography					Encephalography		
	Pos	Probl	Neg	Vas cular	Non vas cular	Pos	Probl	Neg	Pos	Probl	Neg
Meningiomas	33	1	—	28	5	33	—	—	5	—	—
Glioblastomas	55	1	—	37	17	53	—	1	12	—	1
Other gliomas	9	—	12	2	19	21	—	—	8	—	—
Nonverified	12	?	12	11	15	21	1	4	5	1	—
Adenomas of the hypophysis	—	—	2	—	—	—	—	—	2	—	—
Craniopharyngiomas	—	—	2	—	—	—	—	—	2	—	—
Cysts	1	—	5	—	5	5	—	—	4	—	—
Metastases	35 (9)	1 (—)	5 (3)	24 (11)	12 (1)	53 (10)	—	1 (2)	5 (—)	— (—)	1 (—)

the a p and p a projections  $2.5 \times 10^5$  pulses and in the others  $3 \times 10^5$  pulses are accumulated. The recording in each projection takes about 2 to 3 minutes. The detector front surface must be adjusted perpendicularly to the orbitomeatal line in the a p projections and parallel to it in the vertex views. The detector must also be adjusted parallel with the midline of the patient in the lateral projections. Serial recordings have also sometimes been performed during the first minute in one projection with an exposure lasting 4 to 5 seconds followed by a further recording 1 to 3 minutes after the injection.

A clearly increased uptake is usually present when the brain scanning is positive. If the increased uptake is doubtful it has been deemed positive only if evident in at least 3 different projections. If present in only two projections it has been classified as possibly positive.

The angiography consisted in a series of films in at least two projections with two films per second during the first 6 seconds and at 1 to 2 seconds intervals during another 6 seconds. The techniques described by LINDGREN (1949) have been used for the encephalography.

**Material.** A total of 1 200 brain scanings have been carried out during a 21 month period in 900 patients, 185 of whom proved to have an intracranial tumour (162 supratentorial and 23 infratentorial) at operation or at autopsy.



Fig 1 *Top* Left carotid angiography. Slight displacement of the pericallosal artery and possibly some arching of its branches in the anterior part of the frontal lobe. *Bottom* Encephalography. The roof of the left anterior horn is slightly depressed. No other abnormality evident.

Operation was for various reasons not performed in 26 patients but the presence of a supratentorial neoplasm was considered certain from the typical changes recorded by either brain scanning or conventional neuroradiologic methods or in most cases both of them. Angiography was carried out in 183 patients and ordinary encephalography in 69 patients.



Fig 2 Brain scan An increased uptake is clearly present in the left frontal lobe near the midline

## Results

The title 'vascular' indicates abnormal tumour circulation manifested as vessels of abnormal appearances, contrast medium accumulation (tumour blush) or early filled draining veins (Table 1). Nonvascular means that the angiographic diagnosis was based only on displacement of vessels. Astrocytomas, oligodendrogliomas and ependymomas, grades 1, 2 and 2-3 have been included in the group 'other gliomas'. Operation was for various reasons not performed in the group 'nonverified tumours' most of which were centrally situated. The diagnosis was considered certain from the evidence of multiple tumours and a known primary growth in a few cases of metastases and not subjected to operation.

The only meningioma that was not obvious at brain scanning was a recurrence situated outside the vault. It should be mentioned in this connection that the interpretation of the scan in a possible recurrence is often somewhat uncertain since an increased uptake may frequently occur for a long time after operation. Angiography was not performed in one patient in whom the conventional encephalography was positive. The accuracy was also good in glioblastomas (Table 1). The only patient in whom both angiography and encephalography were negative had a 'probable positive uptake at brain scanning'. Four months later a typical glioblastoma was present when the angiography was repeated. A glioblastoma of the frontal lobe in another patient was somewhat more precisely localized at brain scanning than at angiography and encephalography (Figs 1, 2).

An increased uptake was recorded in less than 50 per cent of the patients in the group 'other gliomas'. In one patient with both solid and cystic tumour parts the brain scanning revealed only the former but the angiography indicated both (Figs 3, 4). Only 2 of the 15 'nonvascular' neoplasms in the 'nonverified' group produced positive brain scanning. A reduced uptake was recorded in the serial exposures during the first minute as well as at 1 to 3 and 45 to 60 minutes in one patient of a large cystic tumour.





Fig 3 Left carotid angiography. Marked displacement of the pericallosal artery to the right, the middle cerebral artery is elevated. Marked stretching of arterial branches in the temporal lobe, as well as in the basal part of the frontal lobe, indicating a growth on both sides of the Sylvian fissure.

The accuracy of brain scanning in detecting metastases is fairly good but it was nevertheless negative in 5 patients whereas angiography and encephalography were negative in only one patient. The growth in 3 patients with a negative brain scanning appeared at angiography to be vascular. Multiple lesions were present in 12 patients; in two revealed at brain scanning but only single 'vascular' tumours at angiography. On the other hand brain scanning was negative in 3 patients in whom angiography demonstrated multiple vascular tumours.

*Infratentorial tumours.* The accuracy of brain scanning was as usual less in these than in supratentorial neoplasms (Table 2). Some improvement was gained if the head was flexed forwards so as to elevate the transverse sinus for the p.a. film. A metastasis not evident at encephalography was detected by angiography, otherwise the former appeared to be the more reliable method.

*Results of serial recordings during the first minute and at 1 to 3 minutes as compared to those at 45 to 60 minutes.* An increased uptake was evident as early as the first minute in 26 out of 36 examinations and at 1 to 3 minutes in 43 out of 45 patients (Table 3). The uptake in most of the meningiomas was more intense during the first minute and at 1 to 3 minutes than at 45 to 60 minutes (Fig. 5). (This has also been observed in 3 patients with arteriovenous malformation.) Somewhat more marked uptake at 1 to 3 minutes than at 45 to 60 minutes was also sometimes evident in metastases and



Fig. 3. Brain scan. Only the ventral part of the tumor and not its entire part in the frontal lobe is evident.

glioblastomas 9 out of 25 patients; but as a rule the uptake was greatest at 45 to 60 minutes. PLANTOL 1954<sup>4</sup> as well as others (HARNA et coll. 1969) was the first to point out the value of registration at different times after the injection. The present authors have had no cases of increased uptake during the first minutes and none at 45 to 60 minutes later. As expected the stronger uptake during the first minutes was usually observed in highly vascular lesions and the two tumors without such uptake during the first 1 to 3 minutes were both poorly vascularized according to angiography. However in 6 vascular tumors (3 meningiomas, 2 glioblastomas and 1 metastasis) a higher uptake was recorded at 1 to 3 minutes although not during the first minute after the injection. Nevertheless the result of this investigation indicates that the increased uptake is partly caused by increased vascularization.

### Conclusion

Conventional neuroradiologic methods obviously possess a degree of accuracy approaching 100 per cent. The reliability of brain scanning is also high in supra-tentorial meningiomas and glioblastomas but somewhat lower in metastases. In other supra-tentorial and in intra-tentorial tumors the accuracy is only about 50 per cent. In all three peritumoral meningiomas investigated however an increased uptake was recorded. The method may on the other hand, be of considerable value in screening a possible neoplasm since no complications arise and little discomfort is caused to the patient. The method may also be of value where, exceptionally the changes apparent in conventional methods are too slight to be clear-cut. Brain scanning is also of great help in predicting the nature of the neoplasm. If the brain scanning is negative a supratentorially located meningioma can almost certainly be ruled out and the presence of a glioblastoma deemed unlikely. Brain scanning may if necessary be repeated several times, which is of particular value in the differential diagnosis between a new growth and

Table 2

*Results of brain scanning angiography and encephalography in 23 patients with intracranial tumours*

	Brain scanning			Angiography					Encephalography		
	Pos	Probl	Neg	Vas cular	Non vas cular	Pos	Probl	Neg	Pos	Probl	Neg
Meningiomas	1	—	1	1	—	1	—	—	2	—	—
Neurinomas	5	—	3	—	2	2	—	—	8	—	—
Astrocytomas (cerebellum)	1	—	—	—	—	—	—	—	1	—	—
Gliomas (medulla oblongata)	—	—	1	—	1	1	—	—	1	—	—
Ependymomas	—	—	1	—	—	—	—	—	1	—	—
Hemangioblastomas (cerebellum)	—	—	1	—	—	—	—	—	1	—	—
Papillomas (of plexus chorioideus)	—	—	2	?	—	2	—	—	1	—	1
Metastases	5	1	1	2	—	2	—	—	5	—	1

Table 3

*Results of serial recordings during the first minutes and at 1 to 3 minutes as compared to those at 45 to 60 minutes after injection*

	Brain scanning					Angiography				
	4 to 5 second serial records				At 1 to 3 min	At 45 to 60 min	Vas cular		Non vas cular	
	Pos	Probl	Neg	Not per formed	Pos	Neg	Pos	Neg		
Meningiomas	14	8	—	3	3	14	—	14	13*	—
Glioblastomas	17	12	—	2	3	17	—	17	10*	6
Other gliomas	3	—	—	3	—	1	2	1	2	3
Nonverified	3	2	—	1	—	3	—	3	2	1
Metastases	8	4	—	1	3	8	—	8	6	—

\* 1 not performed

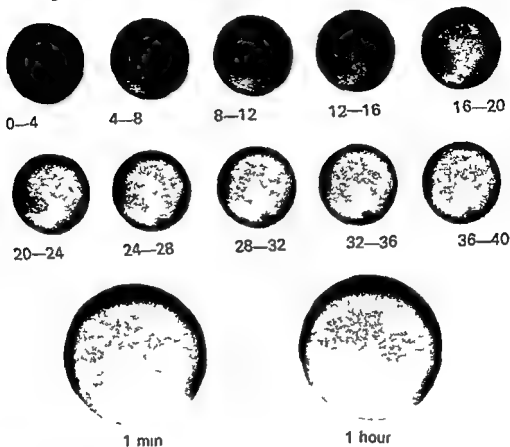


Fig 5 Serial brain scanning in a patient with a meningioma. The increased uptake is intense as early as about 90 seconds as well as at 1 to 3 minutes but weaker at 45 to 60 minutes following the injection.

cerebrovascular lesion (This will be further discussed in another paper, Bo WALLIUS *et coll*.) It may well be that systematically performed recordings at different times after the injection will further increase the possibility of differentiating between the types of neoplasms.

### SUMMARY

The diagnostic accuracy of conventional methods of neuroradiologic investigation and brain scanning have been compared in a large material over nearly a two-year period. The former possess an accuracy approaching 100 per cent. Brain scanning is also of considerable value in certain conditions particularly as it may be repeated if necessary several times.

## ZUSAMMENFASSUNG

Die diagnostische Genauigkeit konventioneller Untersuchungsmethoden und diejenige der Gehirnszintigraphie wurde an einem grossen Material während beinahe einer zwei Jahres Periode verglichen. Die ersten besitzen eine Genauigkeit die an 100 Prozent heran reicht. Die Gehirnszintigraphie ist unter bestimmten Bedingungen auch von bedeutendem Wert, besonders da sie falls notwendig mehrmals wiederholt werden kann.

## RESUME

Les auteurs ont compare l'exactitude diagnostique des methodes classiques d'examen et de scintigraphie cerebrale sur une grande serie couvrant une periode de presque 2 ans. Les methodes classiques ont une exactitude approchant cent pour cent. La scintigraphie cerebrale presente aussi un interet considerable dans certaines affections, en particulier parce qu'elle peut etre repetee si il le faut plusieurs fois.

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FROM THE DEPARTMENTS OF NEUROLOGY (DIRECTOR PROF T BROMAN), RADIATION PHYSICS (DIRECTOR ASSOC PROF H SKOLDBORN) AND DIAGNOSTIC RADIOLOGY II (DIRECTOR PROF I WICKBOM), SAHLGREN'SKA SJUKHUSET GÖTHEN-  
BURG SWEDEN

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## BRAIN SCANNING WITH GAMMA CAMERA AND ANGIOGRAPHY IN CEREBROVASCULAR LESIONS

by

M BOWALLIUS A LARSSON H SKOLDBORN and I WICKBOM

The diagnosis of a cerebrovascular lesion is primarily based on the sudden onset of signs arising from one hemisphere or from the brain stem. It is on the other hand often impossible to determine from such signs alone whether they are caused by an intracerebral hematoma or by infarction. Since the treatment is different in these two conditions a correct diagnosis is essential. Furthermore a tumour may also occasionally give rise to a rapid onset of signs. The correct diagnosis can often be obtained with the aid of cerebral angiography. It has been demonstrated that an increased uptake at brain scanning may occur not only in tumours but also in cerebrovascular lesions (Glasgow et coll 1965). The accuracy and diagnostic value of these two methods has been investigated in a material of proven or possible cerebrovascular hemispheric lesions in which brain scanning was performed during the period 1 March to 31 December 1969.

*Methods* The techniques employed are described in another paper in this issue (Bowallius et coll). The scanning was performed with a gamma camera after the administration of  $\text{Na } ^{99\text{m}}\text{TcO}_4$ .

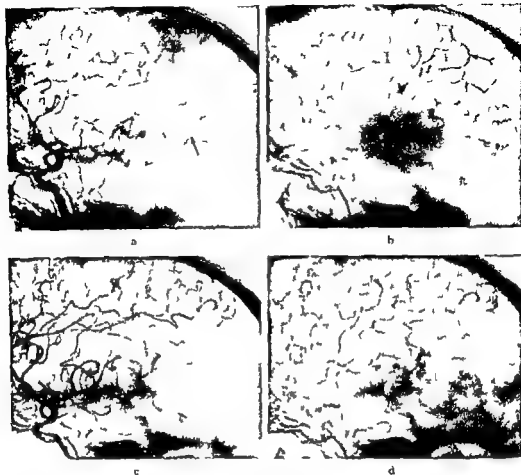


Fig. 1 Left carotid angiography. Male aged 68 with dysphasia, alevia, acalculia, apraxia and right sided hemiparesis of sudden onset but increasing for some weeks. Sudden accentuation of signs at 11 months. a) b) Examination one month after onset of symptoms. Increased vascularization and an early filled vein ( $\rightarrow$ ) at the upper part of the central sulcus. c) d) One month later. No obvious hypervascularization or early venous filling but one branch of the middle cerebral artery, earlier apparent ( $\rightarrow$ ) is not filled. The corresponding part of the parietal lobe appears almost devoid of vessels. (Left carotid angiography 7 months later revealed occlusion of the internal carotid artery.)

**Material.** This consisted of 77 patients scanned once or several times within two months following the onset of symptoms. Carotid angiography was performed in 55 and aortocervical angiography in 13 of these patients. The final diagnosis in 15 patients was intracerebral haematoma, in 2 probable haematoma, in 21 infarction and in 32 patients probable infarction. Seven patients with arteriovenous malformations have all been included since these had a sudden onset of

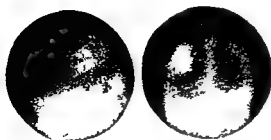


Fig 2 Same case as in fig 1 Left lateral and p a scans one month after first signs. Wedge shaped area of increased uptake superficially in the left parietal lobe. (Four months later further scanning was normal)

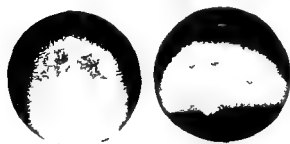


Fig 3 A p and right lateral scans in a right intracerebral haematoma.

symptoms as well. The diagnoses were usually based on clinical signs in combination with laboratory findings and changes evident at brain scanning and angiography since pathologic confirmation could be obtained only in some few patients. An intracerebral haematoma was confirmed in 2 patients at operation and in 2 patients at autopsy. The diagnosis in the remaining 11 patients was based on the demonstration of a space-occupying lesion at angiography in combination in one patient with extravasation of contrast medium and in 10 patients with evidence of subarachnoid bleeding (blood stained CSF yellow after centrifugation and a positive pigment analysis). The diagnosis in one of 2 patients registered as probable haematoma rested on the demonstration of a fairly large space occupying lesion one day after symptoms commenced and in the other on yellow coloured CSF a few weeks after their onset. The former patient with a positive scanning had normal CSF and in the latter angiography was not performed.

Infarction was confirmed at autopsy in 2 patients. This diagnosis in the remaining patients was suggested by (1) angiographic evidence of arterial occlusion with or without retrograde contrast filling of the peripheral branches and abnormal vascularization (Figs 1, 2) (15 patients) (2) repeated attacks indicating transient ischaemia in the same region later becoming manifest (3 patients) (3) bilateral signs from the occipital lobes of sudden and simultaneous appearance (one patient).



Table 1

*Results of brain scanning in 77 patients with hemispheric cerebrovascular lesions*

	Pos	Probl	Neg
Haematoma	10	1	4
Probable haematoma	1	—	1
Infarction	15	—	6
Probable infarction	7	1	24
Arteriovenous malformation	4	1	2

Table 2

*Results of carotid angiography in 55 patients with hemispheric cerebrovascular lesions*

	Pos	Probl	Neg
Haematoma	11	—	—
Probable haematoma	1	—	—
Infarction	16	—	2
Probable infarction	9	2	7
Arteriovenous malformation	7	—	—

If none of the criteria mentioned could be demonstrated in patients with more or less sudden hemisigns this diagnosis was probable infarction (32 patients). Angiographic abnormalities if any in this group were displacement of vessels, usually slight indicating a poorly vascularized lesion or local changes in the circulation with somewhat early venous filling or slow disappearance of the contrast medium from small arteries in one area.

## Results

*Haematoma and probable haematoma* (Tables 1 and 2) Repeated scanning was performed in 6 of the 11 patients with an increased uptake. In 2 of these the investigation was first negative then positive and later gradually returned to negative. The maximum uptake in one patient occurred 16 days and in the other 2 to 5 months after the onset of symptoms. The examinations in the remaining 4 patients did not cover the whole course. An increased uptake was still evident in one patient two years after the onset of symptoms. The area of increased uptake was with two exceptions less distinct than in the patients with infarction and somewhat more irregular in shape. It could be located either

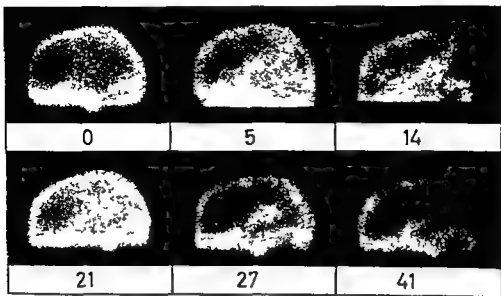


Fig 4 Repeated left lateral scanning 0 5 14 21 27 and 41 days after start of symptoms in cerebral infarction. Area of increased uptake in the left temporo-parietal region with typical features

superficially or more centrally (Fig 3). In 4 of the 5 patients in whom the examination was negative the recording was performed within 12 days of the first symptoms.

*Infarction and probable infarction* (Tables 1 and 2) Repeated examinations were performed in 21 of 23 patients with positive scanning. In 3 of them the whole course could be followed with a normal first examination and later with an increased uptake which eventually disappeared, the maximum uptake occurred between the 7th and 30th day after the start of symptoms (Fig 4). In 15 patients at least one of the recordings was performed when the uptake was most marked during this period (7th to 30th day) which will be called the optimal time for brain scanning. This increased uptake was still evident more than two months following the first symptoms in 4 patients. The area of increased uptake was usually wedge shaped (Figs 2-4) but sometimes rounded with a less marked centre (Fig 5); it was always situated superficially in the area supplied by the middle or the posterior cerebral artery (Fig 2). Five of the patients with a negative scan were examined before and 4 patients after the optimal period. Twenty-one examinations were normal despite the fact that they were performed during this period. Most of these belonged to the group probable infarction.

Table 3

*Comparison between brain scanning and carotid angiography in patients with intracerebral haematomas*

	Carotid angiography			
	Pos	Probl	Neg	Not performed
Scanning				
Pos	7	—	—	3
Probl	—	—	—	1
Neg	4	—	—	—

Table 4

*Comparison between brain scanning and carotid angiography in patients with infarctions*

	Carotid angiography			
	Pos	Probl	Neg	Not performed
Scanning				
Pos	10	—	2	3
Probl	—	—	—	—
Neg	6	—	—	—

Table 5

*Comparison between brain scanning and carotid angiography in patients with probable infarctions*

	Carotid angiography			
	Pos	Probl	Neg	Not performed
Scanning				
Pos	4	1	1	1
Probl	—	—	—	1
Neg	5	1	1	12

Angiography was negative in 11 out of 11 patients in whom it was performed more than two weeks after the onset of symptoms. Since angiographic changes in arterial occlusion may disappear within a short time (GANNON & CHALT 1962, DALAL *et coll.* 1965, ZATZ *et coll.* 1965) this possibility cannot be excluded. The first two weeks will be called the optimal time for angiography. Four patients with transient ischaemic attacks are not included in the material, the brain scanning was negative in all 4 patients.

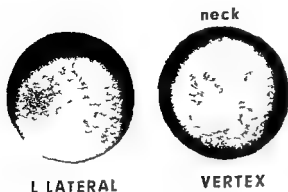


Fig 5 Scanning in cerebral infarction in the left parietal region. Area of increased uptake rounded and its centre less intense

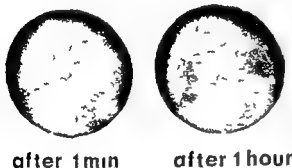


Fig 6 Right lateral serial scans in an arteriovenous malformation. The process is outlined as early as 12 to 16 seconds and more marked at 1 to 3 than at 45 to 60 minutes

*Comparison between brain scanning and angiography* (Tables 3, 4 and 5). A discrepancy between the two methods in 18 patients (4 with haematomas, 11 with infarctions and 3 patients with probable infarctions) was evident. In 11 of these only one of the methods was performed during the optimal period (scanning within 12 days in the 4 patients with haematomas). In the remaining 7 patients who underwent both examinations at the optimal time, the scanning was negative. It should however be pointed out that angiographic changes

were included in the criteria for the diagnosis and also that the angiographic changes were sometimes vague. The former was performed more than two weeks following the observation of symptoms in the 3 patients in whom the angiography was negative and the scanning positive. This suggests that the isotope examination may be more reliable if the patient for some reason is examined more than two weeks after the stroke. Another advantage of brain scanning is that repeated examinations may be performed without any risk to the patient. This may be of help in the differential diagnosis between a cerebrovascular lesion and a tumour, especially when the symptoms develop more gradually. The isotope examination may also be of value where the angiographic changes are somewhat questionable.

An intense uptake was evident in 4 patients with arteriovenous malformations (Fig. 6). The malformations were small in the 2 patients with a negative brain scan (Table 1).

*Serial recordings during the first minutes.* These were performed in 1 patient with haematoma, in 7 with infarction and in 3 patients with probable infarction, in all of whom an increased uptake was evident 45 to 60 minutes after injection. In only one of them was the increased uptake present during the first minute and in 4 patients after 1 to 3 minutes, although less marked than at 45 to 60 minutes (ECONOMOS *et coll.* 1966; HANCA *et coll.* 1969). This indicates that the increased uptake depends on damage to the blood-brain barrier rather than on increased vascularization.

The process was much more obvious during that time than at one hour in the 3 patients with arteriovenous malformation in which serial 4 to 5 s exposure recordings were made during the first minute (Fig. 6) (SCHLESINGER *et coll.* 1962; HANCA *et coll.* 1969).

## SUMMARY

The diagnostic accuracy of angiography and brain scans in cerebrovascular lesions has been investigated in 77 patients. The lesion could be demonstrated more often at angiography than at brain scanning. However, the results are very much depending on the time interval between the onset of symptoms and the examination. For a brain scan the optimal time appears to be 4 to 6 weeks, whereas angiography preferably should be performed as soon as possible and at least during the first two weeks. Scanning may be helpful in the differentiation between haematoma and infarction and should, if positive, be repeated later in order to rule out neoplasia.

## ZUSAMMENFASSUNG

Die diagnostische Richtigkeit der Angiographie und der von Gehirnszintigrammen bei cerebrovaskulären Schaden wurde bei 77 Patienten untersucht. Die Schaden konnten

häufiger durch die Angiographie als die Gehirnszintigraphie nachgewiesen werden. Jedoch sind die Ergebnisse sehr stark vom Zeitintervall zwischen dem Einsetzen der Symptome und der Untersuchung abhängig. Der optimale Zeitpunkt für die Gehirnszintigraphie scheint 4 bis 6 Wochen zu sein, während die Angiographie möglichst unmittelbar und wennstens innerhalb der ersten zwei Wochen ausgeführt werden sollte. Das Szintigramm mag bei der Differentialdiagnose zwischen einem Hämatom und einem Infarkt anwendbar sein und sollte wenn es positiv gewesen ist, später wiederholt werden, um ein Neoplasma auszuschließen.

## RÉSUMÉ

Les auteurs ont étudié la précision diagnostique de l'angiographie et des scintigraphies cérébrales sur 77 malades atteints de lésion cérébro-vasculaire. La lésion a pu être mise en évidence plus souvent par l'angiographie que par scintigraphie. Cependant les résultats dépendent beaucoup de l'intervalle de temps entre le début des symptômes et l'examen. Pour une scintigraphie cérébrale le temps optimal paraît être de 4 à 6 semaines alors que l'angiographie devrait de préférence être faite aussitôt que possible ou au moins au cours des deux premières semaines. La scintigraphie peut être utile dans le diagnostic différentiel entre hématome et ramollissement et devrait dans les cas où elle est positive être répétée plus tard de façon à éliminer une tumeur.

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## CEREBRAL SCINTIPHOTOS WITH $^{113m}\text{In}$

by

A. CONSTANTINOVICI

The application in practice of scintigraphy with short lived radionuclides produced by laboratory generators has considerably advanced this diagnostic technique during the last few years. It is now possible to perform cerebral cinthphotography with  $^{99m}\text{Tc}$  which irradiates the organism a hundred to a thousand times less than, for instance  $^{201}\text{Hg}$  (BECK et coll 1967, WITCORSKI et coll 1967). Moreover, the short radioactive half life of these radioisotopes permits repetition of the examination at much shorter intervals than with  $^{131}\text{I}$  compounds and may likewise be considered an important step forward.

The following short life isotopes are at present given preference in cerebral photoscanning  $^{99m}\text{Tc}$ ,  $^{67}\text{Ga}$  and more recently  $^{113}\text{In}$ , to these may be added  $^{18}\text{F}$ , produced in the cyclotron, and  $^{197}\text{Hg}$  both with shorter half lives but not produced by laboratory generators. In spite of the particular difficulties encountered and the poor retention of radionuclides by the cerebral tissue the possible simultaneous demonstration of all the brain regions by scintiphotography with a scintillation camera is the second considerable stride made in examination of the brain with radioisotopes during the last years (ANGER & ROSENTHAL 1959).

The introduction into practice of a generator of  $^{99m}\text{Tc}$  from  $^{99}\text{Mo}$  (HARPER et coll 1962) and of  $^{67}\text{Ga}$  from  $^6\text{Ge}$  (GREENE & TUCKER 1961, YANO & ANGER

1964) was followed by that of a generator of  $^{113m}\text{In}$  from  $^{113}\text{Sn}$ , suggested by the investigations of GREENE et coll (1963) and later of STANG & RICHARDS (1964) and first used in clinical practice in 1967 (SUBRAMANIAN & McAFFEE)

The physical properties of  $^{113m}\text{In}$  and of its parent have been discussed in many papers (ADATEPE et coll 1968 O MARA et coll 1969, SUBRAMANIAN & McAFFEE 1967) The half life of 99.3 minutes, almost four times shorter than that of  $^{99m}\text{Tc}$  and its harder monoenergy radiation of 393 keV are important Among the factors with a biologic effect is the half life of 1.6 h for 80 per cent and somewhat longer for the remaining 15 per cent of the half life The radiation of the organism is almost double that of  $^{99m}\text{Tc}$  (O MARA et coll), the critical organs being both the large intestine and the urinary bladder, the elution of  $^{113m}\text{In}$  from the generator may also present a certain danger due to its energy spectrum and great affinity for bone Contamination with  $^{113m}\text{In}$  usually however does not exceed  $10^{-5}$  to  $10^{-6}$

The most important advantage of  $^{113m}\text{In}$  over  $^{99m}\text{Tc}$  is the physical half life of the  $^{113}\text{Sn}$  generator (118 d), much longer than that of  $^{99}\text{Mo}$  the intervals at which the source must be renewed are thus decreased although the price of indium lies in the possibility of repeating elution of the source at 8 h instead of at 24 h intervals as with technetium Indium gives a clearer craniocerebral impression because its concentration at the level of the salivary glands and choroid plexuses is lower than that of technetium and its clearance from the blood more rapid These advantages are however, more theoretic than otherwise, especially with the nonchelated form, indium chloride

Another drawback of indium compared to technetium is its rapidly decreasing activity that limits the duration of the procedure and the more hazy character of the craniocerebral representation, this is due to the much higher proportion of diffused radiation that penetrates through the collimator insufficiently thick for 393 keV The demonstration of cerebral tumours is much improved however by complexing the indium eluate with various chelating agents of which DTPA has produced the best results (STERN et coll) The tumour/brain uptake ratio has given some authors in experimental investigations a ratio twofold better with  $^{113m}\text{In}$ -DTPA than with  $^{99m}\text{TcO}_4$  (29.3 as against 14.6) (O MARA et coll) Similar investigations have been carried out with various complexed forms (DTPA, EDTA HEDTA NTA, tetracycline, citric acid) which yielded 1.5 better results than the eluate used as such (BURDINE et coll 1968)

*Material and Method* A total of 120 patients with various neurosurgical conditions received 10 mCi of  $^{113m}\text{In}$  eluted from an  $^{113}\text{Sn}$  column (Amersham) with 5 ml of 0.05 N HCl Ten other patients received 10 ml of the complexed forms prepared as follows (1) With diethylenetriaminedipentaacetic acid (DTPA),



Table 1

*Results in various lesions*

	Correct findings	Incorrect findings	Total
Meningioma	11 (91.7 %)	1 (8.3 %)	12
Glioblastoma	7 (77.8 %)	2 (12.2 %)	9
Metastasis	9 (75 %)	3 (25 %)	12
Acoustic neuroma	2 (40 %)	3 (60 %)	5
Other supratentorial tumours	15 (62.5 %)	9 (37.5 %)	24
Other infratentorial tumours	6 (54.5 %)	5 (45.5 %)	11
Hematoma (extracerebral)	1 (70 %)	4 (30 %)	5
Cerebral circulatory lesions (incl. angiomas)	10 (83.3 %)	2 (16.7 %)	12
Intraorbital non tumoural processes	2 (66.7 %)	1 (33.3 %)	3
Non neurosurgical lesions (incl. functional disturbances)	21 (91.3 %)	2 (8.7 %)	23
Technical errors	—	4	4
Total	84 (70 %)	36 (30 %)	120

Table 2

*Results in various locations*

	Correct findings	Incorrect findings	Total
Cerebral hemispheres	33 (71.7 %)	13 (28.3 %)	46
Medial and paramedial supratentorial lesions	7 (77.8 %)	2 (12.2 %)	9
Posterior fossa (lateral)	10 (66.7 %)	5 (33.3 %)	15
Posterior fossa (medial)	3 (50 %)	3 (50 %)	6
Parasellar location	—	1	1
Pterion location	1 (50 %)	1 (50 %)	2
Orbital and retroorbital lesions	2 (66.7 %)	1 (33.3 %)	3
Diffuse lesions	19 (86.4 %)	3 (13.6 %)	22
Functional disturbances	9 (75 %)	3 (25 %)	12
Technical errors	—	4	4
Total	84 (70 %)	36 (30 %)	120

1 mg  $\text{Cl-Fe}$  and 2.5 mg DTPA were added to 5 ml eluate. The pH was adjusted to 7 with a buffer phosphate solution (CEA France). (2) With ethylenediamine tetraacetic acid (EDTA), 6 mg  $\text{FeCl}_3$  and 1 ml of a 10% calcium chelate

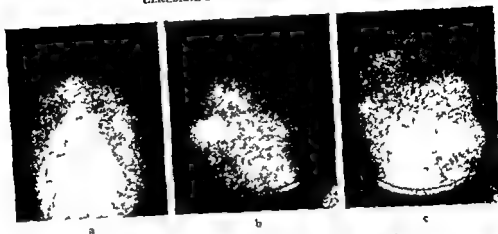


Fig 1 Olfactory meningioma a) Ap b) left lateral and c) right lateral view

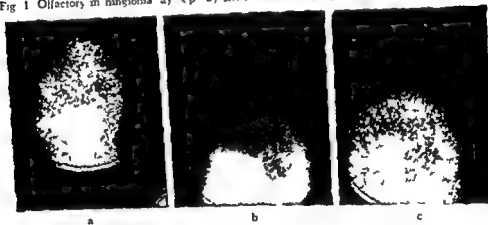


Fig 2 Appearances of left posterior fossa with a)  $^{113m}\text{In}$  chloride b)  $^{99m}\text{TcO}_4$  and c)  $^{99m}\text{TcO}_4$  Neohydriin

solution of EDTA di sodium salt were added to 5 ml eluate. The pH was adjusted to 7 with 2.4 ml of a N 10 NaOH solution (approximately 10 mg). The usual dose was 10 mCi and the number of registered pulses 200 k. dots.

The patients had various neurosurgical and neurologic diseases. Tables 1 and 2 indicate the accuracy of the information supplied by the investigations carried out with  $^{113m}\text{In}$ . Table 1 giving the results in terms of the pathoanatomical lesions and Table 2 its location.

The tables indicate that the degree of accuracy of the information supplied by photo scans with  $^{113m}\text{In}$  depends primarily upon the degree of vascularization of the pathologic process and alteration of the hemato-encephalic barrier at the level. This gives to the method its high degree of sensitivity in meningiomas, glioblastomas and certain localized (angiomas) or diffuse vascular processes. The

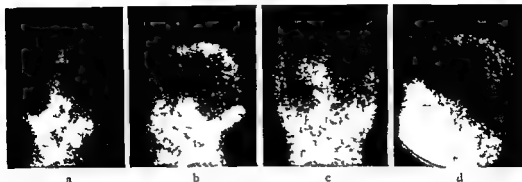


Fig 3 Left parietal metastases a) Pa and b) left lateral view with indium chloride c) Pa and d) left lateral view with indium DTPA

results were far less correct in extracerebral hematomas and in a series of cerebral tumours, such as astrocytomas, oligodendrogliomas and cystic tumours.

The situation of the process exercises almost the same influence. The most difficult lesions to detect were those lying in the posterior fossa, especially along the midline, those situated in the region of the pterion (when even a meningioma could not be differentiated from the hyperactive area of the overlying temporal fossa), and the parasellar ones. Of equal value from the standpoint of the neurosurgical diagnosis were the negative results that eliminated a possible intracranial space occupying process.

### Discussion

The high quality of the scintiphotographic information obtained with  $^{113}\text{In}$  chloride is confirmed by the demonstration of an olfactory meningioma (Fig 1). The comparative value of scintiphotography with indium chloride and with  $^{99}\text{Tc O}_4$  or  $^{203}\text{Hg}$  Neohydrin in a tumour of the left posterior fossa is also evident (Fig 2). Proof of the possibility of obtaining net improvement of the quality of the films with the mixed forms of  $^{113\text{m}}\text{In}$  is supplied by Figs 3, 4 and 5.

The last photoscans demonstrate that in meningiomas the results obtained with  $^{113\text{m}}\text{In}$  chloride are slightly superior in intensity and resolution to those produced by  $^{113\text{m}}\text{In}$  EDTA probably due to the vascular structures particularly abundant in this tumoural variety, and inherent to the oral region. In contrast, the high film quality obtained with the chelate form in the other tumoural varieties indicate a superior uptake of this complex especially in the extracellular space.

### Conclusion

$^{113}\text{In}$  is a useful radionuclide for the photoscintigraphic diagnosis of neurosurgical cerebral conditions as the examination may be performed at a short

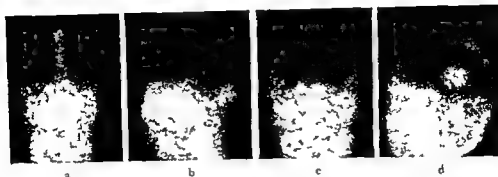


Fig 4 Left temporal glioblastoma a) Ap and b) left lateral view with indium chloride c) Ap and d) left lateral view with indium DTPA

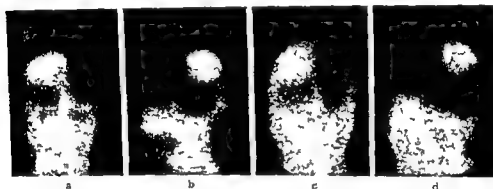


Fig 5 Left parietal meningioma a) Pa and b) left lateral view with indium chloride c) Pa and d) left lateral view with indium EDTA

interval after its intravenous administration. The presence of comparatively strong monoenergy gamma radiation lowers the resolution of detail in the absence of a more powerful collimator than the one used for  $^{99m}\text{Tc}$ .

The film quality obtained and hence its value depends to a great extent upon the chemical form of administration. The author feels that  $^{99m}\text{Tc}$  must be preferred as a routine radionuclide for cerebral scintiphography. However for the sake of economy and rhythmic function of the unit, it is necessary to have both  $^{113m}\text{In}$  and  $^{99m}\text{Tc}$  provided the possibility of chelating the eluate exists.

## SUMMARY

The introduction into practice of cerebral photoscanning with  $^{113m}\text{In}$  has proved extremely useful owing to the physical properties of indium as a source. The information supplied is however inferior to that furnished with  $^{99m}\text{TcO}_4$ . The results obtained with indium chloride in 170 patients and with the forms complexed with DTPA in a further 10 patients demonstrated the necessity of its administration in such forms.

## /USAMMI NI ASSUNC

Die Einführung von  $^{113}\text{In}$  in die Praxis der cerebralen Photoscanning hat sich wegen der physikalischen Eigenschaften von Indium als besonders anwendbar erweisen. Die Information daraus ist jedoch derjenigen die mit  $^{99}\text{Tc O}_4$  erhalten wird unterlegen. Die Ergebnisse die mit Indium Chlorid bei 120 Patienten erhalten wurden zeigten jedoch die Notwendigkeit diese in solchen Formen zu verwenden.

## RÉSUMÉ

L'introduction dans la pratique de la scintigraphie cérébrale par le  $^{113}\text{In}$  s'est montrée très utile en raison des propriétés physiques de l'indium comme source de rayonnement. Cependant l'information obtenue est inférieure à celle que donne le  $^{99}\text{TcO}_4$ . Les résultats obtenus avec le chlorure d'indium chez 120 malades et avec les formes combinées à l'ADTP chez 10 autres malades a montré la nécessité de son administration sous ces formes.

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## BRAIN SCANNING IN CEREBROVASCULAR LESIONS

by

S CROQVIST and R MULLER

Increasing experience with brain scanning has revealed that abnormalities may be encountered in a variety of cerebral lesions apart from brain tumours. Large series in which the result of isotope examinations has been related to the type of cerebrovascular lesion and to the clinical signs have been reported by ECONOMOS (1968) and recently by MARSHALL & POPHAM (1970). A valuable contribution to the discussion on the cause for the isotope uptake in these types of lesions has been made by QUINN (1968). In spite of the information now available many aspects that are incompletely verified and that need further explanation still exist. This report on the results of brain scanning in a series of 172 patients with the clinical diagnosis of a cerebrovascular lesion deals with the following matters: (1) the reliability of the method, (2) the importance of the time interval between the onset of symptoms and the isotope examination, (3) the length of a positive state, (4) the diagnostic importance of a variation in the activity said to occur, (5) the possibility of a diagnosis of the type of cerebrovascular lesion being obtained, and (6) the relation between the results obtained by the isotope examination and angiography.

The relation between the clinical state and the findings at the scanning needs clarification; this also applies to the differentiation between a cerebrovascular lesion and cerebral growth. These aspects will be dealt with in subsequent reports (CROQVIST & MULLER 1972; CROQVIST *et al.* 1972).

*Methods* Brain scanning with a gamma camera has been employed for some time as a routine in combination with cerebral angiography and conventional encephalography, so that a constant comparison and correlation between the methods has been possible. An injection of 10 mCi technetium  $99^m$  pertechnetate was made intravenously about one hour before which 400 mg potassium perchlorate are given to decrease excretion of technetium in the choroid plexus and the salivary glands, atropine is also subcutaneously injected in a dose of 0.5 to 0.75 mg depending upon the age of the patient to decrease salivation. The registration of the isotope activity on a roentgen film by means of a photoscope produces a 1:1 image; this facilitates direct comparison between the scan and other neuroradiologic procedures. The registrations are made at about two and a half to three hours after the injection of the isotope. Five views are taken: both laterals, one a.p., one p.a. and one vertex view. Only appropriate views are obtained when findings have been previously positive and when the examination is repeated. Selective angiography is performed with serial films in both lateral and frontal projections. Alteration of the a.p.c.o. or arterial blood pressure has not been made in relation to the angiography in this series.

The intensity of activity was evaluated in comparison with that of the sagittal sinus at the level of the vertex in each positive examination. Activity equal to that of sinus was marked (+ + +), when above (+ + + +) and when slightly below (+ +). One (+) was the rule when slight but definite isotope was evident. Consideration was further paid to the form, site and demarcation of the area of increased activity, the distribution of the activity within the lesion was also considered. The size of the area with increased activity was calculated from the lateral films in about 50 per cent of the positive findings.

*Material* The series comprises 172 patients, most of whom were 70 years of age or over. Only one isotope examination was made in 89 of these while in the remaining 83 two or more were performed at varying intervals after the onset of symptoms. Two investigations were thus performed in 52 patients: 3 in 22, 4 in 7 and 5 in 1 and 6 in 1. The diagnoses were based upon history, neurologic examinations and the results of chemical and cytologic examinations of the spinal fluid (SÖRNÄS 1967; SÖRNÄS & MÜLLER 1967) but not upon the roentgenologic findings. The conditions consisted of transitory ischaemia clearing within 24 hours in 5 patients, a brain stem lesion in 6, supratentorial infarction and an intracerebral haematoma in 135 and 18 patients respectively. It was impossible to differentiate between infarction and bleeding in 11 patients. The findings in the spinal fluid made it possible further to separate 130 of the 135 infarct patients into two groups consisting of 70 with haemorrhagic and 60 with non-haemorrhagic infarction. It should be stressed that subdural haematoma, subarachnoid haemorrhage or arteriovenous malformations have not been included.

Table 1

*Overall results of brain scanning in supratentorial lesions in relation to clinical diagnosis*

Clinical diagnosis	No of patients	Positive scanning	
		No	Per cent
Transient ischaemic attack	5	1	
Infarction	135	88	65
Haemorrhagic	70	57	81
Non haemorrhagic	60	23	38
Not classified		3	
Intracerebral haematoma	18	16	(89)
Not classified to type	8	6	(75)
Total	166	111	68

The patients have further been divided according to the severity of the signs at the time of the isotope examination. Group 0 includes patients without neurologic signs and group I those in whom they were slight but definite in group II the signs were considered to be of moderate severity while in group III they were advanced. Angiography was performed in 72 patients, 69 of whom were selectively examined.

### Results

A total of 111 positives or 64 per cent, occurred in the complete series of 172 patients. None of the 6 patients with a brain stem lesion was positive, one of the 5 with transitory ischaemic attack was, however positive. Only those patients with signs from supratentorial structures and with a definite diagnosis as to type of lesion will be considered in the following.

About 65 per cent of the patients with cerebral infarction had a positive brain scan. The separation of the infarcts into haemorrhagic and non haemorrhagic types disclosed a notably different result (Table 1). Thus 81 per cent positives were recorded in haemorrhagic as compared to 38 per cent in non haemorrhagic infarction. 16 out of 18 patients with an intracerebral haematoma were positive. The figures are based upon the overall results, if only the initial examinations are considered the figures are somewhat lower.

The results of the initial scanning have been related to the interval between the onset of symptoms and signs and the isotope examination in order to estimate their dependence on time. Of the total number of patients with



Table 2

*Results of brain scanning related to interval between onset of signs and initial examination*

Clinical diagnosis	1 to 7 days		7 days or more	
	No. of patients	Positive scanning (per cent)	No. of patients	Positive scanning (per cent)
Haemorrhagic infarct	45	76	95	92
Non haemorrhagic infarct	27	44	33	33
Intracerebral haematoma	11	(64)	7	(86)
Total	83	64	65	61

haemorrhagic infarct with non haemorrhagic infarct and with haematoma examined during the first week 59 per cent were positive on the first three days 62 on the fourth and fifth day and 72 per cent on the sixth and seventh day. The total percentage positive during the first week was 64, which is about the same obtained in those patients examined at later dates, i.e. 61 per cent (Table 2). Some differences were observed in the results in the three types of lesions. Of the patients with haemorrhagic infarction 76 per cent were positive in the first week and 92 per cent later. The corresponding figures for non haemorrhagic infarction were 44 and 33 per cent. The differences are not larger than might be explained by chance. The group of 18 patients with intracerebral haematoma consisted of 11 examined during the first week with a positive finding in 7, the scanning was abnormal in 6 of the 7 patients examined later.

The patients with repeat examinations were analysed in order further to clarify the influence of the interval between the stroke and the isotope examination (Table 3). A total of 77 patients consisted of 22 who were negative and 55 patients who were positive. The examination in the two groups was made on an average at 4.2 and 7.1 days after the onset of signs respectively. The difference in itself suggests the importance of the time interval and was further strengthened by the fact that repeated examinations disclosed that no less than 8 of the 22 patients initially negative had become positive.

The activity in the lesion varied with time. Twenty-two of the 55 initially positive patients had an increase and 14 a decrease while 19 patients remained unchanged (Table 3). The intensity of the activity reached a maximum in the second and third week followed by a decrease. This pattern was the same irrespective of the type of lesion. Variations in time in the area of abnormal activity are not only confined to the intensity but applied also to the size,

Table 3

*Results at initial examination related to interval from onset of disease and related to findings at repeat examination*

No of patients	Days after onset of signs (average)	Initial scanning	Repeat scanning		
			Unchanged	Increased	Decreased
22	4.2	Neg	14	8	—
55	7.1	Pos	19	22	14

Table 4

*Differential diagnosis from brain scan as to type of lesion*

Clinical diagnosis	No of patients	Scanning diagnosis			
		Haemorrhagic infarct	Non haemorrhagic infarct	Intra-cerebral haematoma	No diagnosis
Haemorrhagic infarct	57	39	3	6	16
Non haemorrhagic infarct	23	2	10	0	11
Intracerebral haematoma	16	5	0	7	4

demarcation and shape. The size thus generally tends to increase, being greatest during the third week while the demarcation towards surrounding tissue becomes more defined. An infarct area initially cone shaped tends with time to become spherical.

Intracerebral haematomas were in most patients located in the central ganglia and consequently best evident in a p.a. and lateral films, sometimes however in the vertex view. The area varied in size but was spherical and fairly well defined; the intensity was evenly distributed and increased with time to reach a maximum in the third week. In cases with infarcts the area with increased isotope uptake was located in relation to the posterior cerebral artery or most often in relation to regions supplied by the middle cerebral artery and its branches. By combining the information from films taken in different projections it was possible to locate the lesion to the larger vessels and even to define the main branch or branches affected. In cases in which the lesion was related to the posterior cerebral artery the abnormal uptake was best evident in lateral and p.a. films; the latter demonstrating the area located close to the mid line. When the lesion was caused by disturbance in the circulation within the middle cerebral artery valid informa-

Table 5  
*Comparison between angiography and brain scanning*

	Selective angiography	Scanning	
		Pos	Neg
Normal	12 (19 %)	3	9
Pathologic	57 (81 %)	42	15
Total	69	45	24

tion was obtained from films taken in the lateral projections and in the a p or in the p a projection depending upon the arterial branches engaged. The activity in both haemorrhagic, and non haemorrhagic infarctions was related to the distribution of the arteries and consequently was more or less cone shaped, i.e. had a triangular appearance both in the lateral and frontal views, with non haemorrhagic infarct the shape was most characteristic and did not markedly change with time. The demarcation of the area with abnormal activity was fairly sharp, the initial intensity being high and increasing in later examinations. The maximum was reached during the third week although it only seldom became as high as in haemorrhagic infarcts or haematoma. No gross variations in activity occurred within the lesion although the size varied. A haemorrhagic infarct presented an isotope uptake with an area that, although having basically a triangular shape, tended to be rounded with irregular poorly defined borderlines. The intensity within the lesion varied, the size of the area was sometimes exceptionally large corresponding to the whole region supplied by the middle cerebral artery. It was in this type of lesion that the most obvious variations in shape, intensity and demarcation occurred with time.

The characteristics described formed the basis for the determination of the type of lesion (Table 4). The appearances in the brain scans were in agreement with the clinical diagnosis in 49 of the 96 patients, in 16 of these a false diagnosis was made and in 31 patients a definite diagnosis was not possible. The activity in the latter was generally low and failed to permit an evaluation of the shape and demarcation. Thirty-two of 41 patients with a haemorrhagic infarct were correctly diagnosed while in non haemorrhagic infarction the figures were 10 out of 12 and with a haematoma 7 out of 12.

Twelve (19 per cent) of the 69 patients in whom selective angiography had been performed were normal and 57 (81 per cent) abnormal (Table 5). The scanning was positive in 3 of the former. One of these was not classified while in the other 2 the diagnosis was haemorrhagic infarction. The angiographic

Table 6

*Angiographic findings related to result of brain scanning*

Main angiographic finding	No of patients	Positive scanning
Normal	12	3
Arterial stenosis	13	6
Arterial occlusion	17	14
Circulatory disturbance*	9	6
Expansivity	18	16
Total	69	45 (61%)

\* Contrast blush early filled veins or collateral circulation without other signs of occlusion

Table 7

*Abnormal angiography negative brain scanning*

Clinical diagnosis	Angiography	Interval onset—scanning	Interval onset—angiogr	Degree of signs
Haemorrhagic infarct	Early filled vein	2	3	0
Transitory ischaemic attack	Early filled vein	6	0	0
Non haemorrhagic infarct	Early filled vein	16	11	II
Non haemorrhagic infarct	Arterial stenosis	8	9	II
Non haemorrhagic infarct	Arterial stenosis	6	1	II
Non haemorrhagic infarct	Stenosis middle cerebral artery	5	11	I
Non haemorrhagic infarct	Stenosis middle cerebral artery	17	16	I
Haemorrhagic infarct	Stenosis internal carotid artery	4	0	II
Non haemorrhagic infarct	Stenosis internal carotid artery	6	10	I
Non haemorrhagic infarct	Stenosis internal carotid artery	0	0	I
Haemorrhagic/non haemorrhagic infarct	Occlusion Sylvian artery	5	5	I
Non haemorrhagic infarct	Occlusion internal carotid artery	26	23	0
Non haemorrhagic infarct	Occlusion pericallosal artery	6	4	III
Non haemorrhagic infarct	Expansivity	16	16	0
Non haemorrhagic infarct	Expansivity	3	1	III

examinations were made on the 9th 20th and 22nd day after the first appearance of signs, respectively. The three isotope examinations were performed on the 13th 14th and 17th day, respectively with an activity of (+) (++) and (+++) in the corresponding patients. The degree of the neurologic signs did not differ from that in the other patients with normal angiographic findings obtained in one patient on the 9th day in the other on the 20th day, and in the third on the 22nd day.

Thirteen of the 57 patients with abnormal angiographic findings (Table 6) had arterial stenosis within the extracranial portion of the internal carotid artery or in the intracranial vessels while 17 had arterial occlusion. Nine patients had circulatory disturbances characterized by contrast blush early filling of veins or of collateral circulation without any other signs of occlusion. Similar changes were present in patients of other groups although they did not constitute the main angiographic findings. An additional 18 patients had angiographic evidence of expansivity most of them had an intracerebral haematoma although expansivity was also present in infarction.

Six of 13 patients with arterial stenosis had a positive scan. Positive results were also obtained in 14 of the 17 patients with arterial occlusion in 6 out of the 9 patients with circulatory disturbances and in 16 of the 18 patients with expansivity. Negative results were recorded in 15 patients with an abnormal angiography. In one of these patients the diagnosis was transient ischaemic attack and in the remainder infarction mostly of the non haemorrhagic type. The patient with transient ischaemic attack and two others had angiographic findings of circulatory disturbances i.e. early venous filling. 7 had arterial stenosis 3 arterial occlusion and in 2 patients signs of expansivity were evident. The degree of the symptoms and signs varied and in comparison with the series was as a whole milder. The interval between their onset and the isotope and the angiographic examinations varied but did not differ from the series as a whole (Table 7).

### Discussion

Most reports on the results of brain scanning in cerebrovascular lesions are based upon examinations performed with mercury 197 chlormerodrine (GLASGOW et coll 1967 ECONOMOS 1968, MARSHALL & POPIAN 1970). Experiences with technetium 99m pertechnetate have also been published (MOLINARI et coll 1967 WITCOWSKI et coll 1967 QUINN 1968). Disagreement over the comparable efficiency of the two radiopharmaceuticals still exists. The properties of  $^{99}\text{Tc}$  appear to be advantageous. The high radiation energy and short physical half line of the tracer permit the use of larger dosage of radio

activity with consequent high counting rate, short recording time and low radiation dose. Comparative investigations in which both compounds have been used have not presented any significant difference in results (QUINN *et coll* 1965 WITCOWSKI *et coll* 1967 CROLL *et coll* 1969). The use of different registration equipment may however influence the results. A gamma camera used in this investigation offers certain advantages in comparison with conventional scanning equipment, the most important being that it allows rapid registrations in readily obtained projections. This possibility has always been utilized in the present series.

The reported incidence of abnormal findings in systematically performed isotope examinations in patients with cerebrovascular lesions varies between 45 and 80 per cent. GLASGOW *et coll* (1967) reported 59 per cent positives, clinically small lesions being excluded. MOLINARI *et coll* (1967) published a figure of 67 per cent while WILLIAMS & BEILER (1966) and ECONOMOS (1968) gave 80 and 79 per cent positives respectively. RHOTON *et coll* (1966) had no positives without residual signs while 25 out of 49 patients with residual neurologic signs were positive. MARSHALL & POPHAM (1970) have made the same observation, only 38 of 103 patients examined were positive, i.e. 37 per cent. Excluding those with transient ischaemic attack 45 per cent of the remaining 82 patients with a completed stroke were positive. Vascular lesions related to the vertebrobasilar system are not demonstrated by the isotope examination unless the posterior cerebral artery is also included. It therefore seems as if the situation of the lesion and the clinical type of stroke may influence the results. Our experiences are partly in accordance with this conclusion. The overall results in our series were 111 positives in 172 patients or 64 per cent, excluding 6 patients clinically diagnosed as having a brain stem lesion the figure becomes 66 per cent. One of 5 patients with transient ischaemic attack had a positive scan. Angiography in this patient failed to reveal any lesion, the only abnormality being localized stenosis in the extra-cranial part of the internal carotid artery. The finding is not in agreement with those made by RHOTON *et coll* or by MARSHALL & POPHAM. Two positives with transient ischaemic attack have however been reported by OJEMANN *et coll*. MARSHALL & POPHAM pointed out that such a result may give rise to doubts about the diagnosis and that malignancy is possible. The course of the disease in the patient in the present series did not, however, suggest a tumour.

According to most observers the time interval between the first appearance of symptoms or signs and the isotope examination markedly affects the frequency of an abnormal uptake, a finding partly confirmed in our series. The number of positives in those patients of the present series in whom the initial examination was performed during the first week was much higher than reported by others.

No less than 64 per cent were thus considered abnormal in comparison to 30 per cent of those mentioned by MARSHALL & POPHAM. It has also been stated that there is only exceptionally an abnormal uptake during the first few days after the *ictus*. In our series no less than 17 of the 29 patients examined on the first to third days were positive. There are probably several explanations for the difference between these figures and those reported by others, most of whom had used a rectilinear scanning device. The great flexibility of the camera permits the technique applied in this series with routine registration in five different projections. The method appears to offer additional advantages since it is the combination of the information obtainable from each registration that yields the best results.

The collimation used may also be of importance in the results. Contrary to the conditions in conventional scanning equipment a gamma camera mainly registers the activity in the superficial structures, the site of most cerebrovascular lesions. The difference in results may further be explained by the fact that the present authors, in contrast to others, have routinely performed the examination at about three hours after the injection of the isotope. Continuous registration of the activity in the blood, unbroken counting of the lesion to non lesion ratio and practical experience have indicated this as being the most appropriate time for registration. The gamma camera and multiple projections together with the choice of an appropriate time after the injection would thus appear to offer better facilities for the early detection of an abnormal uptake.

The fact that the size and activity of a lesion also vary with time may certainly influence the results. Most positive registrations are observed during the second and third week, i.e. the time at which the maximum in size and activity are reached, thus making the lesion more readily demonstrated.

The result of the brain scanning has been related to the type of the lesion in several reports. ECONOMOS reported differences in the frequency of positives in patients with infarcts and haematomas of 79 and 90 per cent, respectively, but also described certain characteristic changes. Only rarely have single patients with infarcts been separated into haemorrhagic and non haemorrhagic types in this series.

The increase in activity with time in all three categories as pointed out by other authors was confirmed. This increase has been suggested to be a diagnostic criterion to differentiate tumours and vascular lesions of the brain. The necessary repeat examinations thus delay the diagnosis. It would appear that the information obtained from one examination usually permits a diagnosis although this is more precise with high activity. The latter is also of importance in determining the type of cerebrovascular lesion from the scanning.

When comparing the results at angiography and isotope examinations cases

demonstrating discrepancies are of most interest. The 3 angiographically normal patients with positive scans were examined late after the onset of signs when minor abnormalities may well have been normalized. General experience has shown that circulatory disturbances may be transitory and confined to the acute phase of the disease.

Fifteen patients in spite of abnormal angiographic findings had negative scans. 7 of these had stenosis of the carotid artery in the neck. Three patients all examined within the first 11 days had early filling veins which although a sign of a localized lesion suggested that only a limited area had been affected. The negative scanning in 5 patients despite arterial occlusion in 3 and an expansivity in 2 patients are more difficult to understand. The signs in one of the latter had been transitory. The patient might be included in the group with reversible ischaemic neurologic signs which, as defined by MARSHALL & POPIAM fails to have an abnormal isotope uptake. The second patient with expansivity was examined only on the third day and it may be that a positive finding would have been encountered at repeat examinations. A patient with occlusion of the carotid artery was examined on the 26th day when no residual signs remained. In a second patient angiography demonstrated occlusion of the pericallosal artery. Due to its central location an abnormal uptake may not have been registered or difficult to discern from the normal high activity in the sagittal sinus. In the third patient with occlusion a small branch of the Sylvian artery was affected and as in early filling veins the region affected might have been only small.

Nine patients of the series had both negative angiography and scanning while in 18 patients one or the other was positive. Abnormalities explaining the signs and symptoms thus occurred together in 60 out of 69 patients. This result is in favour of regarding the two methods as complementary.

### Conclusion

Brain scanning has been performed in 172 cases with cerebrovascular lesion. Using a gamma camera registration the cranial activity was registered 2.5–3 hours after intravenous administration of 100 mCi  $^{99}\text{Tc}^m$  pertechnetate. Registration was routinely made in five different projections. Sixty nine cases were examined one, 83 two or more times.

The clinical diagnosis was that of a brain stem lesion in six cases. In 166 cases with supratentorial lesion the diagnosis was transient ischaemic attack (5 cases), intracerebral haematoma (18 cases) and infarct (135 cases). Abnormal isotope uptake was encountered in 111 cases (64 per cent). All six cases with brain stem lesion were negative. One out of the five cases with transient ischaemic attack was positive. Intracerebral haematomas were positive in 89 per cent, infarcts in



65 per cent. When separating the infarcts in non haemorrhagic and haemorrhagic a marked difference in distribution of positive results was noted, being 81 and 38 per cent, respectively.

With regard to intensity, size, form and demarcation the area with abnormal activity differed in cases with intracerebral haematoma, haemorrhagic infarct and non haemorrhagic infarct. These differences in characteristics make a differential diagnosis as to type of lesion possible from the brain scan. In this series no difference in frequency of abnormal findings occurred between examinations performed during the first week or later. Repeat examinations, however, confirmed the dependence of the result in the individual case upon interval between onset of symptoms and signs and time of scanning.

In 69 of the 166 cases with signs of supratentorial lesion carotid angiography was performed. In three out of 13 angiographically normal cases the brain scan was positive. Out of 57 cases with angiographic changes 15 had a normal scan. Negative result at both angiography and brain scanning was found in only nine cases indicating that the two methods are complementary.

### SUMMARY

A material of 172 patients has been examined by means of brain scanning with a gamma camera. The results obtained are discussed with regard to (1) the reliability of the method, (2) the importance of the time interval between the onset of symptoms or signs and the isotope examination, (3) the length of a positive state, (4) the diagnostic importance of a variation in the activity said to occur, (5) the possibility of a diagnosis of the type of cerebrovascular lesion being obtained and (6) the relation between the results obtained by the isotope examination and angiography. Comparison with results obtained at carotid angiography in sixty nine of 166 patients with supratentorial lesions indicates that the two methods are complementary.

### ZUSAMMENFASSUNG

Ein Material von 172 Patienten wurden mit Hilfe von Gehirnschanning mit einer Gamma Kamera untersucht. Die erhaltenen Ergebnisse werden hinsichtlich folgender Punkte diskutiert: 1. der Anwendbarkeit der Methode, 2. der Bedeutung des Zeitintervalls zwischen dem Einsetzen der Symptome oder Zeichen und der Isotopenuntersuchung, 3. der Dauer des positiven Zustands, 4. der diagnostischen Bedeutung einer Variation in der Aktivität die aufgetreten sein soll, 5. der Möglichkeit einer Diagnose des Typus der cerebro vaskulären Läsion die erhalten wurde und 6. der Relation zwischen den Ergebnissen die mit der Isotopenuntersuchung und der Angiographie erhalten worden waren. Der Vergleich mit den Ergebnissen die bei neunundsechzig von 166 Patienten mit einer supratentorialen Läsion bei der Carotisangiographie erhalten worden waren deuten darauf hin dass die beiden Methoden einander ergänzen.

### RÉSUMÉ

Une série de 172 malades ont été examinés par scintigraphie cérébrale avec une gamma caméra. Les résultats obtenus sont étudiés du point de vue (1) de la fidélité de cette

methode (2) de l'importance de l'intervalle de temps entre le debut des signes fonctionnels ou des signes physiques et l'examen isotopique (3) de la longueur d'un etat positif (4) de l'importance diagnostique d'une variation dans l'activite (5) de la possibilite de faire le diagnostic du type de lesion cerebro-vasculaire et (6) de la comparaison entre les resultats obtenus par l'examen isotopique et l'angiographie. La comparaison avec les resultats obtenus par angiographie carotidienne chez 69 des 166 malades atteints de lesions sus tentorielles montre que ces deux methodes sont complementaires.

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## RELATIONSHIP BETWEEN THE CLINICAL STATE AND BRAIN SCANNING IN CEREBRO VASCULAR LESIONS

by

S CRONQVIST and R MULLER

Experiences with brain scanning in cerebrovascular lesions have indicated that the examination may be normal or present only slight abnormalities even in patients with severe neurologic signs. On the other hand patients with minor signs may have a marked isotope uptake (ECONOMOS 1968; MARSHALL & POPLHAM 1970; QUINN 1968; STEVNER et coll 1968). Since it is mainly the clinical state that in practical work will determine if and when an isotope examination should be performed, it is imperative to know the relation between the two. This relation has been investigated in patients with cerebrovascular lesions with special attention paid to the site and size of the lesion as well as to the activity within it. The question whether brain scanning contains information with regard to the prognosis has further been considered.

*Material and Methods* Eighty nine of a total material of 161 patients were examined at different times after the onset of symptoms. Based upon the histories, neurologic examinations, cerebral angiographies and the results of chemical and cytologic examinations of the spinal fluid (SORNAS 1967; SORNAS & MULLER 1967) the cases were diagnosed as haemorrhagic or non haemorrhagic infarcts or intracerebral haematoma.

Table 1  
*Results of brain scanning*

Clinical diagnosis	No of patients	Positive scanning	
		No	Per cent
Haemorrhagic infarct	70	57	81
Non haemorrhagic infarct	60	23	38
Intracerebral haematoma	18	16	(89)
Non classified	13	9	(73)
Total	161	105	68

Table 2  
*Results of brain scanning related to severity of signs*

Signs	No of patients	Positive scanning	
		No	Per cent
0	15	2	(13)
I	68	46	67
II	25	16	64
III	53	46	86

The signs were determined at the time of each isotope examination and depending upon the severity were rated as 0 to III. Group 0 consisted of patients with no symptoms or signs at the time and group I comprised those with slight but definite signs. Patients with more marked signs were included in group II while those with advanced neurologic signs were confined to group III.

A gamma camera was utilized for the registration of the cranial activity. Films were obtained in five different projections — right and left lateral, anterior and a vertex view. Only the projections best demonstrating the lesion were chosen at a repeat examination when the initial examination had been positive. The registrations started 2.5 to 3 hours after the intravenous administration of 10 mCi technetium 99m pertechnetate. The activity was estimated in comparison with that of the sagittal sinus at the vertex when the isotope uptake was abnormal. It was referred to as (+++) when equal to that at the vertex, as (++) and (++) when below and above respectively, and finally in cases with only a slight but definite uptake (+). The area of abnormal uptake was measured from lateral films in all positives.

## Results

The 161 patients all had neurologic signs lasting more than 24 hours and referable to supratentorial structures. The clinical diagnosis and the examination results are listed in Table 1. Positive findings were obtained in 68 per cent of patients with a marked difference in frequency of the positives between the haemorrhagic and non haemorrhagic types of lesion of 81 and 38 per cent, respectively ( $p < 0.01$ ). The overall results in relation to clinical signs are listed in Table 2. Of the 15 patients in whom at the time of the examination no clinical signs persisted, 2 had positive scans. Patients with signs rated as I, II and III had positives in a frequency of 67, 64 and 86 per cent respectively. A statistically significant difference was thus evident between those without and those with signs ( $p < 0.01$ ) and among the last mentioned, between the most advanced conditions and the others ( $p < 0.01$ ).

The clinical signs are not necessarily stationary in patients with a stroke but may vary with time. No significant relation appears however to exist between the clinical course and the findings at repeat examinations. Thirty-eight of the 89 patients repeatedly examined were selected at random and separated into two groups. The initial neurologic signs did not differ much in these groups. The first group included 20 patients in whom the clinical signs during the follow up time decreased. 14 of these had an initial positive finding. Two of the remaining 6 patients became positive later while four remained negative. The second group comprised 18 patients who did not improve or even deteriorated. 13 were initially positive. 3 later became positive while 2 remained negative. These findings are in accordance with those by USHER & QUINN (1969). A certain difference between the degree of the clinical signs and the results at repeat examination is however evident if only the negative patients are considered. Three of the 5 patients who later became positive had initial signs rated as III. None of the 6 who remained negative was referred to group III although 4 patients were placed in group I. It thus seems as if the tendency to become positive is mainly confined to patients with more advanced initial signs.

*Site.* Most patients clinically diagnosed as having infarcts had symptoms or signs from regions supplied by the middle cerebral artery. The findings at brain scanning were compatible with such a location and it was often possible to define which main branch of the middle cerebral artery that was or had been affected. It was however generally impossible to predict the findings at scanning from the neurologic signs, except when these mainly consisted of impressive aphasia or isolated homonymous hemianopia. Abnormal isotope uptake was then evident in the superficial part of the postero-inferior parietal region in the

Table 3

*Mean area with increased activity related to signs*

	Severity of neurologic signs			
	0	I	II	III
Area (cm <sup>2</sup> )	12.3	14.1	18.1	25.2

Table 4

*Relation between clinical state and result at repeat brain scanning*

Clinical state	No. of patients	Repeat results		
		No change	Decrease	Increase
Equal or worse	18	9	2	7
Improved	20	7	4	9

dominant hemisphere and in areas supplied by the posterior cerebral artery, i.e. close to the midline, in the parieto-occipital regions respectively.

*Size.* A comparison between the clinical signs and the size of a lesion as measured from lateral films demonstrated that with increasing neurologic signs the size became larger (Table 3). Since in an earlier report (CROQVIST & MULLER) the possibility of making a diagnosis from the isotope examination as to type of lesion was emphasized it should be pointed out that the average size of the lesion was largest in haemorrhagic infarct, i.e. the type of lesion that together with intracerebral haematoma presented the most advanced clinical signs. The size was further related to the time interval between onset of the condition and the isotope examination. The area with increased uptake was then observed to become larger with time with a peak in the third week.

*Activity.* The degree of damage to the tissue within the lesion is supposedly also of relevance in the development of clinical signs in addition to the size and type of a lesion. The type and degree of damage will probably decide the amount of radioactive substance in the lesion and thus also the local activity or uptake as evident at scanning.

The activity was investigated in relation to the degree of the neurologic signs in the initial positives in this series. This proved to be equally distributed in patients rated as I and in those rated as II and III, about 40 per cent being (+) 36 (++) and 24 (+++) or (++++) in the respective groups. No

correlation between the isotope uptake and the severity of initial neurologic signs was apparent.

Repeat examinations of cerebrovascular lesions have indicated that the activity within a lesion may vary with time in accordance with the interval between the stroke and date of scanning. All patients in whom more than one scanning had been performed were therefore examined to determine whether such changes in activity are of any prognostic value. The patients were separated into two groups, those with improvement and those who did not improve or deteriorate. The intensity of activity at each investigation was related to the time interval. Both groups contained patients with increased as well as decreased activity. The activity in 20 patients with improved symptoms was unchanged in 7, while 4 had a decrease and 9 patients an increase. The corresponding figures in the other group without improvement were 9, 2 and 7, respectively, (Table 4). Variations in activity may obviously occur without relation to changes in the clinical state. However, in patients in whom improvement occurred the maximum activity tended to be reached earlier than in the other group and the subsequent decrease in activity also appeared to start earlier and to be more marked than in those with no improvement.

Disturbance of consciousness was almost always combined with advanced neurologic signs. A high activity at the initial scanning in these patients tended to increase and to persist for a longer time.

### Conclusion

Brain scanning is positive in about 70 per cent of patients with cerebrovascular lesions in the supratentorial structures with symptoms or signs lasting more than 24 hours. A correlation with the severity of neurologic signs exists. Patients with no symptoms or signs at the time of the isotope examination were seldom positive (2 out of 15 patients). The highest frequency of positives was encountered in those with the most advanced neurologic signs, such patients when initially negative also later tended to become positive more often than others.

The size of the lesion was inclined to become larger with increasing severity of clinical signs but was also related to time, being largest in the third week after the stroke. Great variations were however evident and the observation has per se no diagnostic implication. No significant correlation seems to exist between the clinical state and the intensity of activity in a lesion, the latter varying with time irrespective of changes in the clinical state of the patient. Repeat examinations disclosed slight differences in the pattern of the isotope uptake in patients with and those without clinical improvement. It would appear therefore that brain

scanning fails to furnish valid information as to the prognosis either when performed as a separate or repeat investigation

## SUMMARY

The significance of the uptake in brain scanning is discussed with special reference to a material of 161 patients. No correlation seems to exist between the clinical state and the activity of a lesion so that it would appear that scanning is of little value in the prognosis.

## ZUSAMMENFASSUNG

Die Bedeutung der Aufnahme bei der Gehirnschannung wird unter besonderer Berücksichtigung eines Materials von 161 Patienten besprochen. Es scheint keine Korrelation zwischen dem klinischen Zustand und der Aktivität des Schadens zu bestehen, sodass die Isotopenuntersuchung von geringen Wert für die Prognose zu sein scheint.

## RÉSUMÉ

Les auteurs étudient l'intérêt de la fixation en scintigraphie cérébrale en se basant sur une série de 161 malades. Il ne semble pas y avoir de corrélation entre l'état clinique et la radioactivité d'une lésion de sorte qu'il semble que la scintigraphie cérébrale a peu de valeur pour le pronostic.

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## BRAIN SCANNING IN THE DIFFERENTIAL DIAGNOSIS OF SUPRATENTORIAL TUMOURS

by

S CROŖVIST H O EFRING and R HUGHES

Isotope encephalography or gamma encephalography is a well established method for the detection of intracranial tumours. The use of radioactive substances for this purpose was introduced by MOORE in 1948. Since then the method has been continuously developed both as regards detection equipment and radiopharmaceuticals. The most important improvements during recent years have been the introduction of a new radioactive tracer  $^{99}\text{Tc}^m$  pertechnetate by HARPER et coll (1964) and the development of the scintillation camera by ANGER (1964).

The gamma camera appears to offer several advantages in comparison with the rectilinear scanner. It has great flexibility with easy facilities for examining the patient in different positions. Furthermore a much shorter time is needed for the recording. This permits serial registrations at short time intervals so that the continuous build up of isotope activity within a tumour may be followed, a dynamic investigation with a refined approach to various differential diagnostic problems is thus made possible. The short exposure time also permits the examination of children and those adults who cannot be kept in position long enough for a conventional scan to be made.

The authors possess the facilities for utilizing the advantages offered by the new developments i.e. isotope examinations of the brain are performed after

the injection of  $^{99}\text{Tc}^m$  and a gamma camera is used for registration of the activity. It should be stressed that it would appear that gamma encephalography is as important a method as angiography and conventional encephalography. They are accordingly all used in combination in the routine diagnostic work. Experiences have initiated the following report on the results in comparison and in combination with other neuroradiologic procedures in a series of cerebral tumours. The possibilities of diagnosing the type of tumour will also be considered with specific attention to the results of an easily applied technique for serial registrations with standard equipment.

**Methods** Gamma encephalography is performed after the intravenous injection of 10 mCi  $^{99}\text{Tc}^m$  pertechnetate. In order to diminish the uptake by the choroid plexus and the salivary glands 400 mg potassium perchlorate are given orally and, to decrease salivation 0.5 to 0.75 mg atropine sulphate is injected subcutaneously one hour before the administration of the isotope. The isotope registrations are performed with a gamma camera (Pho-gamma III, Nuclear Chicago). The recordings are usually made three to four hours after the injection of the isotope. The ratemeter is preset at 400 000 counts which are generally reached within four to five minutes. Both lateral views together with a frontal, an occipital and a vertex view are obtained as a routine.

In cases in which a tumour has been demonstrated in previous roentgenologic or isotope examinations or in which the clinical probability of a tumour is almost certain, serial registrations are made immediately after the isotope injection and at 2, 10 and 30 minutes. The projection expected to give the best definition of the lesion is chosen for these films. The routine views are finally taken at three to four hours. The recordings are made by means of a photoscope and presented in roentgen film to give a 1:1 size image of the skull with good facilities for comparison with conventional roentgenologic investigations. Polaroid films are also available.

Selective internal or external carotid injections are usually attempted for the angiographic examination either by direct puncture or with a catheter via the femoral artery. The angiography includes serial films covering 14 seconds in the lateral and 10 seconds in the frontal projection. The encephalographic investigations are made with fractionated injections of oxygen by the lumbar route. The equipment (Mimer III, Elema Schönder) enables a control to be made of the injection of gas under fluoroscopy and linear tomograms to be obtained when the ordinary films do not provide enough information.

**Material** This consisted of a consecutive series of 102 cases with expansive supratentorial lesions. histologic verification of a brain tumour was obtained in 79 of the cases. A puncture was made of an intracerebral cyst but no specimen

the 3 cases with a negative scan. One growth was located in the posterior part of the frontal lobe and some veins were filled early at angiography. In the second case the neoplasm was situated in the corpus callosum with tumour vessels evident in the angiogram. The third glioma, not revealed by gamma encephalography, had a normal encephalography. No angiography was carried out because the symptoms did not justify further investigations. Three months later, however, the patient developed papilledema. Angiography immediately before an emergency operation disclosed an avascular mass in the central part of the frontal lobe. No repeat scanning was performed.

Histologic verification of metastatic lesions was obtained in all 12 cases. No operation was performed in cases with multiple metastatic lesions; these usually appeared at isotope encephalography more sharply demarcated and had a higher intensity than the gliomas but less marked than the meningiomas. The intensity within the masses was however not always the same when more than one was present. Marked differences were thus noted in three cases with multiple lesions. The roentgenologic findings as well as those at gamma encephalography were not in agreement in all of the cases. This will be further commented upon later.

The cases with an oligodendroglioma and an ependymoma both had positive scans. No means of differentiating these types of tumours from other intracranial tumours in the series existed. The case of reticulum cell sarcoma had two lesions both disclosed by isotope encephalography. Neither of the two pituitary chromophobe adenomas in this series were diagnosed at the isotope examination although encephalography revealed marked suprasellar spread.

In addition to the cases referred to above with histologic verification the nature of 22 cases was not confirmed at operation. Only one of these cases had a negative scan. Few signs were evident and the operation was postponed despite the roentgenologic demonstration of an avascular tumour at angiography and encephalography.

The intensity within the tumours has already been mentioned. It was homogeneous in all of the meningiomas but in some of the other tumours it had an uneven distribution. The intensity in all the tumours was estimated in relation to that of the sagittal sinus as described above, i.e. an arbitrary uptake ratio was used. The results are summarized in Table 2. The number of growths in this table exceeds that given in Table 1, since there were as mentioned several cases with multiple lesions. Ten out of 22 meningiomas had an uptake ratio of  $(+++)$  in the rest of the cases it was  $(+++)$ . There was no meningioma with an uptake of only  $(+)$ . The uptake ratio was  $(+)$  in 3 of the 5 astrocytomas and in the other two it was  $(++)$ . In 5 out of 32 malignant gliomas the uptake was  $(+)$ , in 20 it was  $(++)$  and in 7 it was  $(+++)$ . Three

Table 2

*Activity in different types of roentgenologically or histologically verified tumours*

Type of tumour	Activity		
	(+)	(++)	(+++)
Meningiomas	0	10	12
Astrocytomas	3	2	0
Gliomas	5	20	7
Metastases	3	9	0
Others	8	17	3

of 17 metastases had an uptake ratio of (+) while 14 had (++) The oligodendroglioma recorded an uptake of (+), the ependymoma (++) and the reticulum cell sarcoma (++)

The histologically and radiologically verified growths were split up into two groups, according to vascularity Vascular tumours were regarded as those in which a contrast blush pathologic newly formed vessels or early filling veins were present at angiography (Table 3) Among the 69 vascular tumours there was an uptake of (+) in 9 cases (++) in 41 cases and (+++) in 19 cases Among the 30 avascular tumours the uptake was (+) in 10 cases, (++) in 17 cases and (+++) in 3 cases

Serial registrations according to the technique described were carried out in 32 cases, comprising 10 meningiomas 1 astrocytoma 13 gliomas 3 metastases 1 ependymoma and 4 histologically unverified cases The recordings were evaluated as regards changes in the size of the area with abnormal activity as well as changes in the activity with time The films taken immediately after the isotope injection indicated that the activity in the normal vascular bed was high sometimes making the evaluation of the growth difficult A high immediate uptake was evident within the tumour in all the 10 meningioma cases as well as in 1 glioma in 1 metastasis, in the only case with ependymoma and in 2 histologically unverified cases The activity remained unchanged or in meningiomas tended to decrease

An increase in activity or size was recorded in 17 of the cases These comprised 1 astrocytoma 12 gliomas 2 metastases and 2 unverified cases in 13 of which there was an increase in both size and activity in 4 cases the intensity but not the size increased One of the 17 cases had multiple lesions proved to be gliomas at autopsy In one of these no increase in size or activity was present while in

Table 3  
*Different types of tumours related to activity*

Type of tumour	Activity in vascular tumours			Activity in avascular tumours		
	(+)	(++)	(+++)	(+)	(++)	(+++)
Meningeomas	0	9	9	0	1	3
Astrocytomas	0	2	0	3	0	0
Gliomas	4	14	7	1	6	0
Metastases	3	4	0	0	5	0
Others	2	12	3	6	5	0
Total	9	41	19	10	17	3

the other 3 cases a change in the appearance was evident. In another, the case of the astrocytoma had no activity in the tumour until four hours after the isotope injection.

A central part with an intensity lower than the rest of the tumour was observed in the first of the serial registrations in 2 of the cases of intracerebral tumours, while the activity in the films obtained at three to four hours was regular. One of these neoplasms was a metastasis; at operation revealed to be cystic. In the second case needle puncture indicated the presence of a cyst; no specimen was obtained for histologic diagnosis.

The results of the roentgenologic examinations have already been partly discussed. Some points of specific importance should however be mentioned. In two of the 97 cases examined by angiography a discrepancy existed between the angiographic findings and the results at other examinations. One case, a centrally located partly necrotic glioma, was considered to be normal at angiography; the diagnosis was made by encephalography and scanning. In the second case angiography failed to demonstrate one of two parietally located metastatic tumours. Isotope encephalography demonstrated a neoplasm with an activity of (+) in one of the hemispheres and one with an activity of (++) in the other; only the latter was revealed by angiography; it was avascular.

Thirty-seven of the 40 cases examined by conventional encephalography were positive. Two of the three cases in which encephalography failed to demonstrate the neoplasm had a negative isotope encephalography as well. One of them, a meningeoma growing en plaque in the middle fossa, was diagnosed by angiography. The other was a glioma located in the posterior part of the frontal lobe and was diagnosed by angiography performed three months after both scanning and conventional encephalography had been performed. The third

Table 4

*Comparison between roentgenologic investigations and isotope encephalography in histologically verified cases*

	Positive	Negative
Roentgen examinations	75 (95 %)	4
Isotope examinations	68 (84 %)	11

case was a parasagittal parietal meningeoma the lateral ventricle on the side of the tumour was widened but no sign of an expansive lesion was evident. This growth was demonstrated by both angiography and gamma encephalography.

Angiography and encephalography in combination permitted a final diagnosis of a lesion in all verified cases in this series. There were, however, 4 cases in which the initial investigations were negative or in which they were not in complete agreement with the gamma encephalography. Thus out of 79 histologically verified cases adequate roentgenologic abnormalities were encountered in 75 i.e. 95 per cent.

### Discussion

Together with  $^{197}\text{Hg}$  chlormerodrine  $^{99}\text{Tc}^m$  pertechnetate is the most frequently used radiopharmaceutic at the moment. The properties of the two differ however and there are advocates for both. In favour of  $^{197}\text{Hg}$  chlormerodrine is its rapid elimination from the blood and consequent low concentration in the soft tissues of the skull to give a high target to non target ratio. The biologic half life in blood for  $^{99}\text{Tc}^m$  pertechnetate is less favourable. On the other hand, this tracer has a short physical half life 6 hours as compared to 65 hours for the mercury compound and a higher radiation energy, 140 keV compared to 77 keV which according to most authors makes it the best radioactive substance currently available. These properties thus permit administration of a large amount of radioactivity with the highest possible counting rates and the shortest possible recording times at the lowest possible radiation dosage. From 10 mCi  $^{99}\text{Tc}^m$  pertechnetate the dose commonly used the total body radiation dosage has been calculated to be only 0.11 rad.

Mercury compounds and  $^{99}\text{Tc}^m$  pertechnetate do not seem to differ with

regard to the uptake within cerebral tumours. In spite of this the detection accuracy for  $^{99}\text{Tc}$  tends to be somewhat higher than that for the mercury tracer. One possible explanation for this is the employment of different detection equipment and detection technique in the various series. The advantages of the gamma camera have already been discussed. The authors would like to emphasize the importance of making it a routine to utilize the facilities of obtaining registrations in several different projections. For determination of the location of deep lesions the vertex view has proved particularly useful.

The interval between the injection of the isotope and registration may also influence the result. The time at which registration is started seems to vary at different centres. Registrations are sometimes started and completed in immediate connection with the administration of the tracer substance, in others a delay of 30 minutes is the rule. Earlier experiences of the authors with RISA has demonstrated that a successive build up of activity occurs in some neoplasms, that is a tumour was sometimes demonstrated better in the later scans. This observation together with investigations on the variation of the activity in the blood with time by TAUBE *et coll.* (1969), suggested that the investigations should be performed at about three hours after the administration of the tracer. Further experiences with serial registration has proved this to be a suitable time.

The accuracy of gamma encephalography in the detection of tumours in the series was 88 per cent. The figures may appear somewhat higher than that of other authors using the same isotope and the same equipment. It must however be kept in mind that the series has been confined to supratentorial tumours. The material is not yet large enough to warrant any conclusions on the value of gamma encephalography in the diagnosis of posterior fossa tumours.

The possibility of detecting and localizing an intracranial neoplasm depends upon several factors. Apart from the size of the lesion the situation seems to be crucial and the histologic type of the tumour is also of importance. These two latter factors can hardly be considered separately. It is the optimal combination of the two which permits a diagnosis from the isotope examination. This is best illustrated in cases of meningiomas. Previous experience has proved this type of tumour to have an exceedingly high isotope uptake, an observation confirmed in the present series. In spite of this 3 cases had a negative finding at the isotope encephalography, all three being located in the basal part of the middle fossa. The failure is explained by the fact that this is a region in which there is superimposition of normal anatomic structures with high isotope activity. A tumour located deep within the brain could also theoretically be overlooked or not appear in the isotope investigation performed with the gamma camera. This may be explained by the fact that the energy of the radiation decreases rapidly with distance and furthermore that the collimation used particularly permits

Table 5

*Roent enologic finding and location of tumours with negative isotope encephalography*

Type of tumour	Location	Angiography	Encephalography
Meningeoma	Petrous bone	+ vascular	—
	middle fossa		
Meningeoma	Middle fossa	+ vascular	+
Meningeoma	Pontine angle with supratent extension	+ vascular	—
Astrocytoma	Temporal lobe superficial	+ avascular	—
Astrocytoma	Corpus callosum	+ vascular	—
Astrocytoma	Frontal central	+ avascular	Second examination 10 months later vascular tumour gamma en cephalographs +
Glioma	Temporal super ficial	+ vascular	—
Glioma	Corpus callosum	+ vascular	+
Glioma	Frontal central		— 3 months later vascu lar expansive mass
Pituitary adenoma	Sella	— avascular	+ Suprasellar spread
Pituitary adenoma	Sella	+ avascular	+ Suprasellar spread

demonstration of the more superficial structures. These circumstances accentuate the need to use multiple projections in every case as recommended.

The fact that the histologic type is also of importance is evident from the series. A low accumulation of the tracer is thus to be expected in highly differentiated intracerebral tumours such as astrocytomas; that is the tumour to non tumour ratio may not be great enough to make it possible to differentiate the lesion from surrounding tissue. Negative isotope examinations occurred in three out of seven astrocytomas in the series. The activity within the four tumours of this type recorded was weak. As regards the three negative isotope examinations in one the tumour was located in the corpus callosum, maybe partly explaining why it was not seen while the other two were lying in the frontal and in the temporal lobes respectively, locations that should be favourable for their demonstration. It is therefore probable that the negative findings are explained by the histology of the process.

The isotope uptake in malignant gliomas is not consistent and also varies from case to case. Out of the 32 neoplasms disclosed by isotope examinations 7 had



an uptake of (+++), 20 of (++) and the remaining 5 growths merely (+). Two examinations were negative. The false negative results cannot be explained by the location, both tumours growing in superficial structures.

The uptake of the radioactive compound within a tumour does not seem to be merely dependent upon the presence of pathologic vessels. Thirty out of 36 vascular tumours had a positive gamma encephalography. The proportion of negative cases with pathologic vessels was however less with only 6 out of 75 tumours. The intensity moreover varied between cases with vascular and avascular tumours it being on an average higher among the former. Thus out of 99 histologically or roentgenologically verified tumours the uptake ratio arbitrarily classified as (+) (++) and (+++) is described above, was 11, 41 and 19, respectively in vascular tumours as compared to 10, 17 and 3 in the avascular growths.

*The determination of the type of lesion by isotope encephalography is dependent upon the location of the area with abnormal activity in relation to normal structures upon the intensity and the distribution of this activity, and finally upon the demarcation of the area with increased activity.*

All meningiomas with a positive isotope examination in the series displayed a high evenly distributed activity within an area that was well demarcated from surrounding tissue. (There were 12(+++) 10(++) and 0(+)). This together with the situation of the area in regions related to structures from which meningioma generally arise made a differential diagnosis possible in almost all cases.

Astrocytomas had a much weaker intensity 0(+++) 2(++) and 3(+), and the demarcation was poor. There was also some variation in intensity within the lesion. This was even more evident in cases of glioma, which generally had an irregular transition to normal tissue. The intensity however tended to be higher 7(+++), 20(++) and 5(+) than that in astrocytomas. In a few gliomas it was equal to that occurring in meningiomas and since they also tended to have well defined margins it was not always possible to differentiate them from meningiomas. The metastatic tumours had an intensity less than that of gliomas 0(+++), 9(++) and 3(+) the demarcation tended to be somewhat sharper. Multiplicity is in favour of the diagnosis of metastases but does not constitute proof. One case of the series had multiple meningiomas one multiple glioma and one multiple reticulum cell sarcoma.

Although a single examination may sometimes permit a definition of the type of lesion it is generally unreliable or not possible. PLAVIOL has demonstrated that the uptake of tracer substance is different in various types of lesions. There even seems to be a certain pattern in the build up of activity with time an observation which may be utilized in the diagnosis. Serial registrations would therefore be

expected to contribute further information. A robot camera was attached to one of the oscilloscopes of the gamma camera and films were taken during the first few minutes after the injection and then repeated at close intervals with final registrations after about three hours. The early repeat films did not appear to have such practical significance as to warrant their use in routine work, the procedure was also time consuming. The technique was therefore simplified in the way applied in this investigation. This makes it possible to follow the successive increase in activity within the lesion as well as changes in size information that appears to be of definite importance in the differential diagnosis.

Cases with meningioma all displayed a high uptake immediately following the injection and there was no change in the size. The activity remained unchanged or tended to decrease. The uptake was very slow in the only astrocytoma examined with serial registrations the tumour not being apparent until four hours after the administration of the isotope. Increase in size or activity was a characteristic finding in all but one of the 13 gliomas examined in this way. Two of 3 cases with metastases had an increase in size and activity. The characteristic uptake of meningiomas was only evident in 5 other cases including one of glioma, one metastasis, one ependymoma and two unverified neoplasms. This suggests that serial registrations are of value in the differential diagnosis.

Another interesting observation made from the serial registrations is the finding of a low activity in the central part of the lesion in two of the cases. At operation proved to be cystic neoplasms. These regions with low activity were present only in early films. Films obtained at three to four hours indicated a homogeneous activity in the tumour. This may have been due to a concentration of activity in the cysts similar to that which may occur in subdural haematomas.

Several reports have been made on the accuracy of gamma encephalography as compared to conventional neuroradiologic examinations. The results are not always in accordance. This may depend upon the fact that the authors are mainly experienced in only one of the methods. The results obtained by those skilled in both kinds of examinations are more consistent. The angiographic examinations in the present series were positive in 98 per cent and the encephalographic investigations in 92 per cent, they thus displayed a high degree of accuracy. In one of the cases with multiple lesions only one of two lesions was evident at angiography. This illustrated the value of isotope encephalography in the demonstration of such lesions. A positive isotope examination will also localize the lesion apart from proving its existence. This advantage should be utilized in those cases in which the only finding at angiography is a displacement of midline structures without an exact localization of the tumour or tumours. It is obvious that the angiographic examination in these cases is inadequate and that further investigations are necessary. The value of isotope encephalography

under these conditions should be remembered. It should be stressed that the difficulties encountered in the roentgenologic diagnosis of slowly growing intracerebral neoplasms are also encountered with isotope encephalography. This is illustrated by the observations in three cases of astrocytoma. Among those with a positive gamma encephalography were two cases with such vague findings at the first isotope and radiologic investigations that surgical intervention was not considered justifiable. Later examinations revealed definite lesions at both types of examinations. Negative gamma encephalography in an additional astrocytoma case was followed by a positive finding 10 months later. An avascular frontal new growth was evident at angiography at the time of the first examination but which at the time of the second investigation had become vascular.

### Conclusions

A detection accuracy of 88 per cent as recorded in the present series proves gamma encephalography to be a valuable method in the diagnosis of supratentorial tumours. The explanation of negative findings is in some cases due to the situation of the tumour. Neoplasms located near the base of the skull or close to the midline are thus more likely to present difficulties in diagnosis. In other cases the reason may be the histology of the tumour, slowly growing intracerebral neoplasms such as astrocytomas being more often negative than other types.

Differentiation of the type of lesion is of practical importance especially when gamma encephalography is used as a screening method. Some indication as to the histology may be obtained. The presence of multiple lesions usually indicates a wide spread condition but the possibility of multiple growths of other types must be kept in mind. A high uptake and a sharp border together indicates a meningeoma. Further information on the differential diagnosis between a meningeoma and a glioma may be obtained by serial registrations. The technique used by the authors is easily performed with standard equipment and seems to produce satisfactory results.

Although conventional radiologic methods have indicated the diagnosis of all the tumours in the material the authors feel that the information afforded by isotope encephalography has often been of decisive importance. The contribution of the method has been especially great in cases with avascular lesions. The planning of surgical intervention demands that the findings from the radiologic and the isotope examinations should be carefully correlated, it must be realized that the methods are complementary rather than competitive.

## SUMMARY

A material of 102 cases with expansive supratentorial lesions was examined by gamma encephalography, angiography and conventional encephalography. A detection accuracy of 88 per cent proved isotope encephalography to be of definite diagnostic value. It is well suited as a screening method but its greatest importance is in combination with conventional neuroradiologic procedures.

## ZUSAMMENFASSUNG

Ein Material von 102 Fällen mit expansiven supratentorialen Schäden wurde durch Gammaencephalographie, Angiographie und konventionelle Encephalographie untersucht. Eine Genauigkeit von 88 Prozent bei der Aufdeckung dieser Schäden zeigte, dass die Encephalographie mit Isotopen von klarem diagnostischen Wert ist. Diese ist als Übersuchungsmethode gut geeignet, deren grosse Bedeutung hat sie aber in Kombination mit konventionellen neuroradiologischen Verfahren.

## RÉSUMÉ

Une série de 102 cas présentant des lésions expansives sus-tentorielles a été examinée par la gamma-encephalographie, l'angiographie et l'encephalographie ordinaire. L'exactitude de détection dans 88 pour cent des cas montre que l'encephalographie isotopique a une valeur diagnostique certaine. Elle convient comme méthode de dépistage mais c'est en association avec les techniques neuroradiologiques habituelles qu'elle présente son plus grand intérêt.

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## SZINTIGRAPHISCHE UNTERSUCHUNGEN MIT $^{99m}$ - TECHNETIUM-PERTECHNETAT BEI ZEREBRALEN INSULTEN

VON

G. F. FULGER und K. SUMMER

Gehirnszintigraphische Untersuchungen wurden an 141 Patienten mit eindeutiger Insultanamnese und klinischer sicher lokalisierbaren neurologischen Herden durchgeführt. Die Einteilung der Patienten erfolgte in 6 Gruppen nach dem Verlauf der Apoplexie.

I Fluchtig fluchtiger Insult mit akut aufgetretener, lokal verbarer Symptomatik, die innerhalb von 3 bis 6 Stunden abklang, keine faßbare Restsymptomatik. ■ Patienten

II Transitorisch transitorischer Insult mit deutlich lokalisierbarer Symptomatik, die 1 bis 3 Tage bestehen blieb und sich entweder vollkommen oder fast vollständig bis auf geringe Spurensymptome zurückbildete. 31 Patienten

III Insult mit Remission apoplektischer Insult mit deutlich lokalisierter Herdsymptomatik, die längere Zeit bestehen blieb und dann eine gute Teilrückbildung insgesamt oder eine vollständige Rückbildung einzelner Symptome zeigte. 33 Patienten

IV Insult ohne Remission: apoplektischer Insult mit lokalisierbarer Symptomatik, die ohne wesentliche Rückbildung bestehen blieb oder Verschlechterung zeigte: 39 Patienten

V Insult, id Exitum: apoplektische Insulte mit deutlicher Herdsymptomatik ohne merkliche Besserung, wie Gruppe IV jedoch mit Exitus an zerebralem Coma oder Sekundärkomplikationen wie Herzversagen, Lungenodem, Lungenembolie oder diabetisches Coma: 24 Patienten

VI Zerebrale Blutungen: 6 Patienten

*Szintigraphische Untersuchungsmethodik:* Alle Patienten wurden mittels rektilinearer Szintigraphie aller 4 Seiten des Schädels nach jeweils 10 mCi  $^{99m}\text{Tc}$  Technetium Perchnetrit untersucht. Untersuchungsbeginn war 10 Minuten nach intravenöser Injektion. Nach Beendigung des vierten Bildes wurde eine Wartezeit von einer halben bis einer Stunde eingehalten, danach wurde die abnorme bzw. die klinisch wichtigste Aufnahme wiederholt. Diese letzte Aufnahme wurde als verzögertes Szintigramm bezeichnet. Für schwache Patienten stand ein Doppelkopf camera zur Verfügung, der die posteriore Aufnahme des Schädels mit dem Patienten in Rückenlage mit Hilfe eines Unterschlupfopfes gestützte.

## Ergebnisse

An 141 Patienten mit zerebralen Insulten wurden 47 negative und 94 positive szintigraphische Befunde erhalten. Die szintigraphischen Untersuchungen wurden zwischen 21 Stunden und 48 Wochen nach dem Insult durchgeführt. Die positiven szintigraphischen Befunde zeigten eine Vielfalt, wie sie bei experimentellen Prozessen nicht zur Beobachtung kommt.

Die folgenden Variationen wurden beobachtet: (A) Multiple, diffus über der ganzen Hemisphäre verteilte, kleine Aktivitätsflecken, gelegentlich in Gruppen, ohne wesentliche Konfluenz und ohne eindeutig lokalisierbare oder lateralisierbare umschriebene Herde (multipel, kleinfleckig, diffus). (B) Ein verschieden großer häufig über handflächengroßer Bezirk mit kleinfleckigen Aktivitätseinlagerungen innerhalb desselben, ohne sichtbare scharfe Abgrenzung, gewöhnlich von unregelmäßiger Form, manchmal mit konfluierenden Gruppen von Aktivitätseinlagerungen mit höherer Intensität (unscharf, unregelmäßig, inhomogen). (C) Ein verschieden großer Bezirk mit homogen verteilter Aktivitätseinlagerung, handkartenartig oder feig geformt mit unscharfer Grenze (unscharf, unregelmäßig, homogen). (D) Ein scharf begrenzter Bezirk mit homogener Aktivitätseinlagerung, glatten Konturen, meist weit in die Tiefe reichend, oft keilförmig gestaltet (scharf, glatt, homogen).

Tabelle

*Korrelation der Intensität der Speicherung mit klinischem Verlauf der Apoplexie Geringe Intensität der Anreicherung vorwiegend bei flüchtigen und transitorischen Insulten Hochgradige Anreicherung vorwiegend bei Insulten ohne Remission*

Insulttyp	Grad der Speicherung				Gesamt
	0	I	II	III	
Flüchtig	8				8
Transitorisch	25	(6)			31
Erweichung mit Remission	9		13	II	33
Erweichung ohne Remission	1	7	18	13	39
Erweichung ad Exitum	3	2	8	11	24
Blutungen	1		2	3	6
Gesamt	47	24	41	29	141

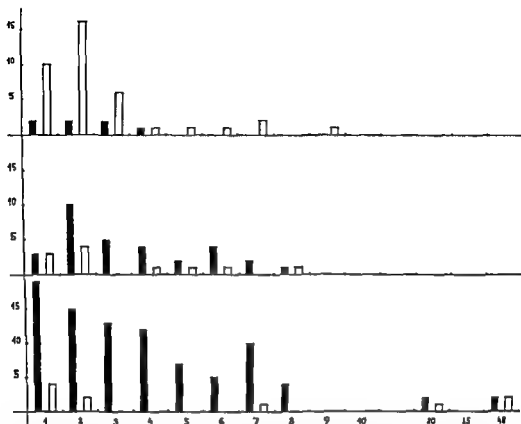
Die szintigraphischen Befunde und die klinischen Gruppen wurden korreliert. Die Diskussion aller untersuchten Beziehungen zwischen Klinik und szintigraphischem Befund überstieg den hier gesetzten Rahmen. Beispielshaft konnte nur die Relation zwischen Intensitätsgrad der Speicherung und Verlauf der klinischen Erscheinungen Erwähnung finden (Tabelle).

Es ergab sich, daß eine hohergradige, zerebrale Schädigung mit schlechter Rückbildungstendenz sehr häufig (90 %) mit einer hochgradigen pathologischen Speicherung des Technetiums einherging, während flüchtige und transitorische Insulte vorwiegend (90 %) mit negativen Szintigrammen assoziiert waren.

Verzögerte Szintigramme lagen in 85 Fällen von den 94 Untersuchungen mit positivem Resultat vor. In 35 Fällen (40 %) nahm die Erkennbarkeit der pathologischen Anreicherung auf dem verzögerten Szintigramm zu; in 10 % der Fälle wurde die umschriebene Läsion erst aus dem verzögerten Szintigramm diagnosifizierbar. In 20 Fällen (24 %) war kein Unterschied in der pathologischen Aktivitätsverteilung des ersten zweiten Szintigrammes der gleichen Seite des Schädels nach derselben Injektion zu beobachten. In 25 Fällen (29 %) nahm die Anreicherung in ihrer Intensität und Erkennbarkeit ab. In 5 Fällen (6 %) war eine Wanderung der Läsion festzustellen.

Neben der Abhängigkeit der Anreicherung vom Intervall zwischen der Injektion und dem Meßzeitpunkt bestand die in der Literatur beschriebene Abhängigkeit vom Intervall seit dem apoplektischen Insult. Die Untersuchung der Beziehung zwischen dem klinischen Verlauf der Erkrankung des Patienten, dem Intervall seit dem Auftreten der Erkrankung und den szintigraphischen Befunden ergab folgendes (Abb.). An den Patienten mit flüchtigen und





Synopsis der Fälle: Schwarze Säulen: Positiver Scan; Weiße Säulen: Negativer Scan. Horizontale Achse: Zeit in Wochen; Vertikale Achse: Anzahl der Fälle. Oberste Reihe: Patienten mit flüchtigen und transitorischen Insulten; Vorherrschend negative szintigraphische Befunde. Mittlere Reihe: Patienten mit apoplektischen Insulten mit guter Remission; Überwiegend positive Befunde von der zweiten Woche an. Unterste Reihe: Patienten mit apoplektischen Insulten ohne Remission oder mit Exitus; Vorherrschend positive szintigraphische Befunde vom Anfang an. Positiver Scan bereits nach 48 Stunden.

transitorischen Insulten waren zu allen Zeiten nach dem Insult kaum pathologische szintigraphische Befunde zu erheben, dagegen entwickelten sich bei Patienten mit Insulten ohne Remission oder mit Blutungen schon frühzeitig und zwar früher als in der Literatur angegeben wurde, pathologische szintigraphische Befunde, auch waren an solchen Patienten nur wenige negative szintigraphische Befunde zu erheben. Die Patienten mit apoplektischen Insulten mit guter Remission nahmen eine Mittelstellung ein und ließen die in der Literatur beschriebene Häufigkeit von pathologischen szintigraphischen Befunden während der zweiten bis vierten Woche erkennen.

Zusammenfassend ergab die Untersuchung von 141 Patienten mit apoplektischen Insulten mittels Gehirnszintigraphie folgendes (1) Die Gehirnszintigraphische Untersuchung mit  $99^m$  Technetium Per technetate ermöglichte die objektive Darstellung geschädigter Gehirnbezirke bei Patienten mit vorangegangenen zerebralen Insulten. (2) Das szintigraphische Erscheinungsbild der pathologischen  $99$  Technetium Anreicherung zeigte erhebliche Variation und ließ sich gewöhnlich auf einen von vier Grundtypen zurückführen (3) Die Übereinstimmung szintigraphisch dargestellter pathologischer Aktivitätsanreicherungen mit der Lokalisation des Herdes im Gehirn war ausgezeichnet (4) Der Intensitätsgrad der Speicherung zeigte ein paralleles Verhalten zum klinischen Verlauf des Insultes (5) Die Gehirnszintigraphischen Befunde hatten auf Grund ihres zeitlichen Auftretens nach dem apoplektischen Insult und auf Grund der Speicherintensität eine prognostische klinische Bedeutung Pathologische szintigraphische Befunde konnten innerhalb von 48 Stunden nach dem Insult auftreten und signalisierten damit eine sehr geringe Wahrscheinlichkeit für eine Remission der neurologischen Ausfälle (6) Zurückbleibende verzögert aufgenommene Szintigramme der verdächtigten oder pathologisch veränderten Seite des Schädels waren eine wertvolle Bereicherung der nuklearmedizinischen Diagnostik zerebraler Läsionen Im Falle von apoplektischen Insulten ermöglichte das verzögerte Szintigramm eine Verbesserung der Diagnostik (7) Durch regional differenzierende quantitative Analyse ergaben sich Einblicke in das biologisch dynamische Verhalten der geschädigten Gewebsbezirke (8) Die Ergebnisse der Untersuchungen erlaubten mit einem hohen Grad von Wahrscheinlichkeit die Unterscheidung zwischen Beeinträchtigung des Funktionsstoffwechsels oder des Strukturstoffwechsels im Rahmen des apoplektischen Insultes

## ZUSAMMENFASSUNG

Technetium Gehirnszintigraphien an 141 Patienten mit zerebrovaskulären Insulten zeigten an 37 von 39 Patienten (87%) mit flüchtigen oder transitorischen Insulten keine Speicherung während 59 von 63 Patienten (94%) mit Insultsymptomatik ohne Remission eine pathologische Technetium Speicherung erkennen ließen. Je intensiver die Speicherung war und umso früher sie auftrat desto geringer war die Wahrscheinlichkeit einer Rückbildung der Insultsymptomatik. Die Gehirnszintigraphie an Insultpatienten ergab die Lokalisation und das Ausmaß der humorganischen Schädigung sowie prognostische Hinweise

## SUMMARY

Cerebral scintigraphy with technetium was performed in 141 patients in whom a cerebral accident had occurred. Thirty two out of 39 patients (87 per cent) with fleeting or transitory signs presented no uptake against 59 out of 63 patients (94 per cent) with chronic

signs and uptake of technetium. The chances of remission of cerebral changes clearly receded with greater and more immediate uptake. Scintigraphy permitted an estimate to be made of the location and extent of the brain damage and helped in the prognosis.

### RÉSUMÉ

La scintigraphie cérébrale au technetium sur 141 malades ayant présenté un accident cerebro vasculaire n'a montré aucune fixation chez 32 malades sur 39 (82 %) qui avaient eu un accident cérébral passager ou transitoire alors que chez 59 malades sur 63 (94 %) qui avaient une symptomatologie d'accident vasculaire cérébral sans remission on a constaté une fixation pathologique du technetium. Plus la fixation était intense et plus elle était précoce moins probable était la disparition sans séquelles de la symptomatologie d'accident vasculaire. La scintigraphie cérébrale chez les malades ayant eu un accident vasculaire cérébral a indiqué la localisation et l'étendue des lésions cérébrales organiques ainsi que des éléments de pronostic.

## ANGIOGRAPHY AND XENON 133 CLEARANCE IN THE INVESTIGATION OF THE BLOOD FLOW OF THE VERTEBRO BASILAR SYSTEM

by

A. JERELL, J. DIEHL and S. ELNAS

Only a limited number of investigations dealing with cerebral blood flow measurements in the vertebro-basilar system has been published. FREYGANG & SOKOLOFF (1958) investigated the regional blood flow in the cat with an autoradiographic method. SKINHØJ et coll (1964) in a clinical material of 7 patients recorded regional blood flow over the cerebellum and the occipital region by the krypton clearance method. external counting was obtained by two scintillation detectors placed on the side of injection. The results presented by these authors are mainly in accordance with those of FREYGANG & SOKOLOFF with higher flow values supratentorially than infratentorially (Table 1). INGVAR et coll (1965) recorded the regional blood flow in the occipital area in 7 healthy young men and reported lower flow values than those of SKINHØJ et coll (Table 1).

*Material and Methods* The material included 19 patients with varying neurologic signs and symptoms examined with the xenon clearance method of LASSEN & INGVAR (1963). An amount of 500  $\mu$ Ci of xenon 133 dissolved in saline was injected into the vertebral artery in conjunction with vertebral

Table 1

*Results of regional cerebral blood flow measurements in ml/100 g/min obtained supratentorially (ST) and infratentorially (IT)*

	Flow of grey matter		Flow of white matter		Mean flow	
	ST	IT	ST	IT	ST	IT
FREYGANG & SOKOLOFF (1958)	12.5	6.9	2.3	2.4		
SKINHOJ et coll (1964)	10.7	6.5	2.9	1.3	5.1	3.3
INGVAR et coll (1965)	80.5		21.1		50.8	
Present investigation	55.0	48.7	19.0	16.9	37.6	32.2

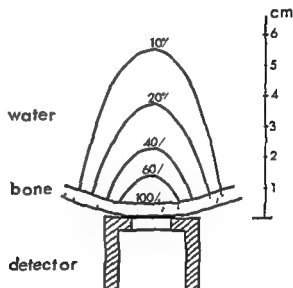


Fig. 1. Isoresponse curves for the type of detector used in the present investigation.

**angiography** The regional uptake was recorded with four scintillation detectors equipped with lead collimators with a depth of 4.5 mm and a diameter of 14 mm. The clearance curves were analysed by the two compartmental method. Isoresponse curves for the type of detector were determined in a phantom in which brain was substituted by water and the vault represented by a piece of calvarium (Fig. 1). Two detectors were placed supratentorially symmetrically over the occipital area in order to avoid contribution from the posterior fossa they were not sited perpendicularly but directed slightly more cephalad to the vault. The two other detectors were positioned infratentorially symmetrically



Fig 2 Position of the scintillation detectors over the occipital area and the cerebellum

behind the mastoid process and directed medially, anteriorly and slightly towards the vertex (Fig 2). To avoid overlapping from supratentorial areas the infratentorial detectors were placed with their centres about 3 cm below the transverse sinus represented by a line drawn from the occipital protuberance to the external meatus. This positioning was later adjusted to bring the centre of the detectors about 2 cm more cephalad.

### Results

The earlier measurements mentioned and the present results are represented in Table 1. The flow values in the present material are calculated as the mean of two symmetric recordings. These flow values, especially those obtained occipitally, are essentially lower than those of SKINHOJ *et coll* and INGVAR *et coll*. This is probably explained by the fact that the present material includes essentially patients with neurologic symptoms and signs justifying vertebral angiography. The values of white matter flow are at variance with those of SKINHOJ *et coll* but in good accord with those of FREYGANG & SOKOLOFF, who reported them to be equally large supra- and infratentorially. The mean flow values recorded supratentorially in the present material did not seem to be

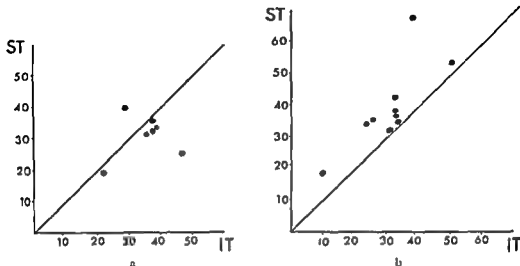


Fig 3 Correlation between the supratentorial (ST) and infratentorial (IT) mean flow values (ml/100 g/min) with high (a) and low (b) positioning of the infratentorial detectors

significantly different from those recorded infratentorially with a high position of the infratentorial detectors (Fig 3a). With low positioning, however, a definite tendency existed for the infratentorial flow values to be lower than the supratentorial values (Fig 3b).

The results indicate that lower flow values should be observed should the detectors be sited too caudally over the cerebellum. The following two experiments were carried out to explore this matter further. Three detectors were placed in a row parasagittally on the side of injection. One detector was located infratentorially in the high position, one in the low position and a third detector was positioned completely below the posterior fossa at the level of C2. The flow values obtained infratentorially in this experiment were in direct relationship to the position of the detector, the mean flow values being 76.9, 25.5 and 12.3 ml/100 g/min. These differences were considerable, the mean flow value recorded over the lower part of the posterior fossa being only a third of that obtained in the high position. In the other experiment four detectors were placed infratentorially, two in the high position, one on each side, and two in the low position, one of the latter detectors being somewhat more caudal than the other. Table 2 indicates that the flow values observed were definitely lower with the caudally situated detectors. This could have been due to regional differences in cerebral blood flow. Wilkinson et al (1969) have pointed out that the grey and white matter vary widely in their relative

Table 2

*Regional blood flow values (ml/100 g/min) recorded with four detectors placed infratentorially two in a high position one on each side and two in a low position. The latter were sited asymmetrically the one on the injected side being somewhat more caudal than the other*

	Injected side				Non injected side			
	High position		Low position		High position		Low position	
Mean flow	39.1		15.5		40.6		30.6	
Fast and slow flow (grey and white matter)	58.3	29.7	55.4	14.2	55.4	26.3	58.3	20.4
Relative weight of grey and white matter	37.9	67.1	13.2	86.6	49.3	50.7	27.0	73.0

amounts in different parts of the brain even in relatively small adjacent areas. This implies that a comparison of right and left side regional flow values will be heavily influenced by the position of the detectors.

### Discussion and Conclusions

These experiments suggested that the infratentorially recorded blood flow is to a great extent dependent on the position of the detector and decreases with a low positioning. This could be due to regional variations in the relative weights of grey and white matter influencing regional flow values (WILKINSON *et al.* 1969). A perhaps more valid explanation would be an extracranial contribution to the clearance curve recorded in the lower occipital area. The vertebral artery indeed, gives off several branches to the muscles of the neck, this being constantly observed in the present material. Although reflux of contrast medium often appeared in the contralateral vertebral artery, this reflux was usually not sufficient to fill the muscular branches. The flow in muscular tissue is known to be comparatively low (LASSEN 1964) in relation to that of brain tissue. With a low positioning of the infratentorial detector the contribution from the muscles of the neck might influence the measurements and result in too low flow values (HOEDT RASMUSSEN 1967). The filling of muscular branches on the contralateral side ought in cerebellar blood flow investigations to be insignificant due to the small volume of xenon injected. A comparison between the flow values on the injected and the non injected side was therefore undertaken. The values were recorded on the two sides with



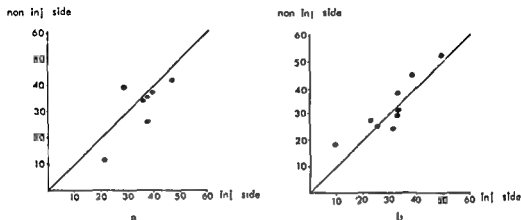


Fig. 1 Comparison between the infratentorial mean flow values (ml/100 g/min) on the injected side and the non injected side the detectors being situated in the high (a) and low (b) positions respectively.

high and low positioning of the infratentorial detectors in the latter case an obvious tendency towards a diminished flow on the injected side existed, most likely due to the extracranial contribution to this side (Fig. 4).

Experience with seemingly correctly placed detectors indicates that cerebral blood flow measurements can be applied in the vertebrobasilar system despite the difficulties in achieving an accurate recording of the infratentorial blood flow. Blood flow measurements and angiography revealed correlation to exist between the regional flow measurements and the regional circulation times as determined by angiography in a patient with metastases in the right cerebellar hemisphere (CROONQVIST & GRITZ 1969, GRITZ 1969). This was true both as regards the relationship between the infratentorial and supratentorial circulation times and to that between the circulation on the affected side and that on the contralateral side (Fig. 5).

The investigation has indicated that the counting geometry is of the utmost importance in posterior fossa blood flow measurements a fact that appears not to have been pointed out earlier. The infratentorial detectors should be placed below the attachment of the tentorium i.e. inferior to a line drawn from the external meatus to the occipital protuberance, but should be positioned as close to this line as possible with due consideration to the dispersion curves of the detector used. The recorded clearance curve will have received a considerable contribution from the muscles of the neck at the level of the foramen magnum. It would appear that a significant risk for incorrect positioning of the infratentorial detectors exists unless their position is checked roentgenologically before the

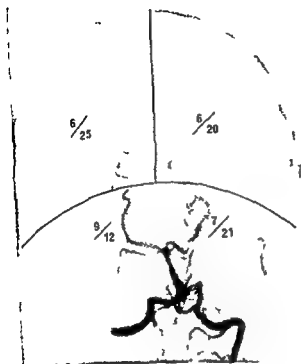


Fig 5 Correlation between the regional circulation time (6, 7 and 9 s) and the regional cerebral blood flow (25, 20, 21 and 12 ml/100 g/min) in a patient with metastases in the right cerebellar hemisphere. The lines indicate the attachment of the tentorium and the superior sagittal sinus.

start of the measurements. It is most likely that further improvements have to be made, possibly by the use of focused detectors and stereotaxic roentgen control to obtain reliable results.

### Acknowledgement

This work was supported by a grant from the Swedish Cancer Society.

### SUMMARY

The regional cerebral blood flow of the vertebrobasilar system has been recorded in 19 patients by the xenon 133 clearance method and intraarterial injection. It is pointed out that meticulous control of the counting geometry is of the utmost importance in avoiding false results from any contribution by the extracranial circulation.

### ZUSAMMENFASSUNG

Die regionale cerebrale Durchblutung des vertebrobasilaren Systems wurde bei 19 Patienten mit der Xenon 133 Clearance Methode und intraarterieller Injektion registriert.

Il wird hervorgehoben dass die minutöse Kontrolle der Messgeometrie von grosser Bedeutung ist um falsche Ergebnisse durch jeglichen Beitrag der extracranialen Zirkulation zu vermeiden

## RÉSUMÉ

Le débit sanguin cérébral régional du système vertébro-basilaire a été mesuré sur 19 malades par la méthode de clearance du Xenon 133 et par injection intra-artinelle. Les auteurs soulignent qu'il est très important de contrôler méticuleusement la géométrie du comptage pour éviter des faux résultats provenant de l'enregistrement de la circulation extracranienne.

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## ULTRASOUND



## OBJECTIVE ECHO ENCEPHALOGRAPHY USING A COMPUTER TECHNIQUE

by

DOLGLAS GORDON

It was at the Fourth Symposium Neuroradiologicum held in London in 1955 that the present writer was the first to describe echo encephalography to a medical audience

The credit for the first observation must however be given to Smith and Turner a radiotherapist and a physicist on the staff of the Royal Cancer Hospital in London who as early as 1948 attempted to use ultrasound to study the effects of radiotherapy on cerebral tumours They discovered however that the effect of the bone of the adult skull was to distort the direction of the beam so much that the method had to be abandoned for this purpose They did however exhibit the apparatus at the Physical Society's Exhibition in London in 1954 and thus showed the possibility of using the technique for diagnosis

The poor efficiency of quartz and the amplifiers available at that time made it very difficult to obtain echoes from the brain and the early paper described only the use on volunteers It did however accurately forecast most of the limitations as well as the uses of the method and it ended as follows First the energy applied must be reduced to a point where no risk to the tissues is involved There are good reasons for believing that this stage has already been reached'

Secondly it is necessary to discover a satisfactory method of introducing the rays at an angle. It is simple enough to introduce the rays at right angles to the skin of the forehead using jelly as a coupling medium. It is however to be hoped that it will be possible to pass rays through the unshaven scalp at angles up to forty five degrees to the perpendicular without having to resort to the immersion of the head in water or to the use of dangerously high power.

Fifteen years later it is only in obstetrics that any lingering doubt remains as to the safety of ultrasonic diagnosis so the first problem has been solved. The second problem has been shown to be impossible to solve. The laws of physics are not capable of being altered by any technologic advance. It has been shown that where media differ in their transmission velocity for ultrasound as much as do soft tissue and bone the angle cannot depart far from the normal or there will be total reflection.

Consequently though probes and amplifiers improved so that the thickest adult skull now yields echoes, it has never been possible to obtain echoes from structures such as the tentorium cerebelli that are inconveniently oriented within the skull.

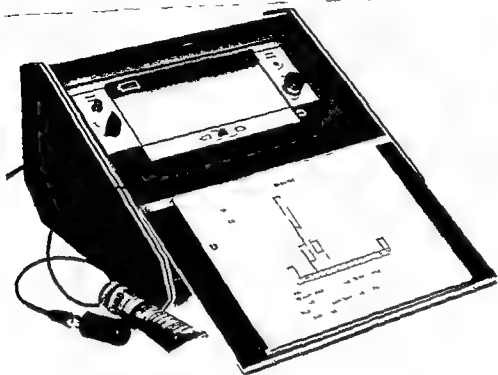
In the 1955 paper the writer warned. The phenomena of reverberation, refraction and diffraction which distort the path of the beam ensure that even in experienced hands interpretation will be difficult. Interested parties ignored this and unjustified claims were made. Longer experience and careful study of the acoustics of skull bone have shown that echo-encephalography's true place is to exclude a displacement of the brain but not to do more than indicate the need for other investigations in patients who have abnormal brain anatomy.

A particular tribute must be paid to WHITE of Kingston, Ontario who more than anyone else investigated every aspect of the effect of bone on the ultrasonic beam and has recently published a monograph on the subject.

The writer has long held the view based on providing a service to a large number of London hospitals that the value of echo encephalography is greatest in the peripheral hospital and least in the fully equipped centre for neurologic diseases where isotope investigations, carotid angiography or pneumo-encephalography involve a long journey for the patient an atraumatic way of selecting the right cases for transfer can save lives.

At peripheral hospitals however it is rare to find the appropriate apparatus and even more so to find anyone with enough experience to make the diagnosis with any confidence. There is also the problem raised by WHITE that an examiner who knows the clinical picture will be tempted to accept as the midline echo that which best fits his preconceptions.

The consequence of this is that echo-encephalography has been condemned by many as too subjective to be relied upon and this is a fair criticism.



The Midliner as opened up for use

To eliminate this subjective element and convert the technique to a scientific objectivity it is necessary to eliminate observer bias. The interpretation must therefore be by the application of rigid criteria, in other words by the use of a computer technique.

As in 1955 the writer must disclaim any originality. The instrument to be described, the Midliner, is the invention of a physicist, J. H. Williams of Lexington, Massachusetts, and the writer's contribution has been minimal. The instrument is in effect the pulser-amplifier of the conventional equipment with a numerical indication of the result of a computer analysis instead of the conventional oscilloscope display which requires human interpretation.

The probe is applied to one side of the patient's head and rocked until an audible signal is heard and one of the seventeen figures on the scale lights up. This reading is recorded on a special chart and a release button is then pressed.

A series of about 20 such readings is obtained quite quickly and the instrument is then switched over for operation from the other side of the head. A further



Secondly it is necessary to discover a satisfactory method of introducing the rays at an angle. It is simple enough to introduce the rays at right angles to the skin of the forehead using jelly as a coupling medium. It is however to be hoped that it will be possible to pass rays through the unshaven scalp at angles up to forty five degrees to the perpendicular without having to resort to the immersion of the head in water or to the use of dangerously high power.

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agreed with the opinion of the writer using the conventional oscilloscope instrument

In elderly patients where a large number of echoes occur in the central region of the brain, but none is significantly larger than the others the instrument gives a random result. This would not cause any incorrect diagnosis to be made and would be the result to be expected if an inexperienced operator were to use the oscilloscope type of instrument.

It must be stressed however that it cannot be used for any other purpose in its present form. The writer has however been engaged in measuring the axial length of the eye to 0.02 mm and has designed a similar instrument to give a direct indication of this measurement. Unfortunately computer techniques do not yet operate at high enough frequencies to achieve high precision but it is already possible to measure to approximately 0.1 mm in theory.

### Conclusions

Echo-encephalography is of greatest value in selecting those cases in peripheral hospitals that should be transferred to a neuroradiologic centre. In such hospitals there is a great need for an instrument that can be operated by inexperienced staff after very short instruction. The Midliner is a very promising application of computer technique to the elimination of the subjective element.

### SUMMARY

Echo-encephalography has been considered too subjective in practice and has been abandoned by some for this reason. The introduction of the Midliner computerised instrument eliminates the subjective element and gives consistent results irrespective of the experience of the operator. Only where multiplicity of echo signals makes interpretation difficult or doubtful does the Midliner give a random result.

### ZUSAMMENFASSUNG

Die Echo-Encephalographie ist als zu subjektiv für die praktische Anwendung beurteilt worden, und ist aus diesem Grund von einigen verworfen worden. Die Einführung eines die Mittellinie berechnenden Instruments schaltet dieses subjektive Moment aus und gibt unabhängig von der Erfahrung des Ausführenden übereinstimmende Ergebnisse. Nur wenn die Vielzahl der Echosignale die Deutung schwierig oder zweifelhaft macht, gibt das Mittellinieninstrument ein zufälliges Ergebnis.

### RÉSUMÉ

Certains auteurs ont considéré l'écho-encephalographie comme trop subjective en pratique et l'ont abandonnée pour cette raison. L'introduction de l'instrument Midliner équipé d'un

calculateur élimine l'élément subjectif et donne des résultats constants indépendamment de l'expérience de l'opérateur. C'est seulement quand la multiplicité des échos rend l'interprétation difficile ou douteuse que le Midliner donne un résultat aléatoire.

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## ECHOENCEPHALOGRAPHIE MIT SIMULTANER A- UND B-BILDDARSTELLUNG

VON

E. KAZNER

Bei der eindimensionalen Echoencephalographie oder A-Bild-Methode werden alle senkrecht zum einfallenden Ultraschallstrahlenbündel stehenden Grenzflächen als Zacken auf dem Bildschirm einer Kathodenstrahlröhre dargestellt. Aus dem Abstand dieser Zacken oder Echos vom Sendepuls ist es bei annähernd gleichbleibender Schallgeschwindigkeit des untersuchten Mediums möglich, die Entfernungen der reflektierenden Grenzflächen vom Prüfkopf zu messen. Sind nur wenige Echos vorhanden, so wird die Interpretation des A-Bildes keine besonderen Schwierigkeiten bereiten. Treten jedoch zahlreiche Zacken auf, dann gelingt eine korrekte Zuordnung der einzelnen Echos zu bestimmten intrakraniellen Strukturen oft nicht mehr.

Diese allgemein bekannten Schwierigkeiten ließen schon lange eine zweidimensionale Darstellung echoencephalographischer Befunde wünschenswert erscheinen, da man sich hiervon eine vereinfachte Lesbarkeit und gleichzeitig mehr Objektivität versprach.

Bei der Ultraschalltomographie oder B-Bild-Methode erscheint an Stelle der Zackenkurve eine Lichtpunktzeile. Die Größe bzw. Helligkeit der einzelnen Lichtpunkte entspricht der reflektierten Ultraschallenergie. Jede Zeile eines zweidimensionalen Echogramms kann man sich als eindimensionales Ultraschallbild vorstellen, das um 90° nach oben gekippt ist, wodurch man gewisser

maßen von oben auf die verschiedenen Echozacken blickt. Ein zweidimensionales Echogramm setzt sich aus zahlreichen derartigen Lichtpunktzeilen zusammen, die durch die Bewegungen des Prüfkopfes in der Abtastebene entstehen und den Eindruck eines Schnittbildes des untersuchten Körperabschnittes vermitteln, etwa vergleichbar mit einem anatomischen Querschnitt.

Ultraschalltomographische Verfahren wurden bereits 1952 von HOWRY & BLISS in die Diagnostik von weichen Körpergeweben eingeführt. Am intakten Schädel wird etwa seit 1963 in Amerika, Europa und Japan mit Ultraschallschnittbild-Verfahren experimentiert. Dabei gelangten zahlreiche Abtasttechniken zur Anwendung (Linear Scanning, Circular Scanning, Sector Scanning, Compound Scanning). Zufriedenstellende Resultate boten sich bisher nur unter Verwendung der Compound Scanning Technik bei Kindern (DE VLIJGER 1968, 1969, VALKEAKARI 1971), während die Ergebnisse der linearen Abtasttechnik (GALICICH et coll. 1965, LOMBROSO & ERBA 1968) eine zu starke Verzerrung aufweisen.

Die eigenen Erfahrungen mit der zweidimensionalen Echoencephalographie reichen bis 1966 zurück. In diesen Jahren haben wir verschiedene Verfahren erprobt, unter anderem auch die Linear Scanning Technik, die jedoch wegen der soeben erwähnten Verzerrung und aus anderen technischen Gründen wieder aufgegeben wurden.

Seit 1969 benutzen wir ein Ultraschallschnittbildgerät, das mit einem Speicherschirm ausgestattet und zur Compound Scanning Technik verwendbar ist. Es enthält ein zweites Oszilloskop für die gleichzeitige A-Bild-Anzeige (Abb. 1). Der Prüfkopf befindet sich am Ende eines Schwenkarmes, der alle in einer Ebene möglichen Bewegungen zuläßt. Diese Bewegungen des Ultraschallstrahlenbündels in der Abtastebene werden als analoge Bewegungen der Zeitbasislinie auf dem Speicherschirm wiedergegeben (Abb. 2). Genaue Lage und Richtung des Prüfkopfes überträgt dabei ein Positionscomputer, dem die Daten von drei Präzisionspotentiometern zugeführt werden.

Während der Ableitung, d. h. während der Prüfung, befindet sich der Prüfkopf in einer bestimmten Schnittebene um den Kopf herum bewegt wird. Erscheint auf dem Speicherschirm sukzessive das B-Bild, auf dem zweiten Oszilloskop stets das augenblickliche eindimensionale Bild. Die Kontrolle durch dieses A-Bild bietet die Möglichkeit, die für den Compound Scan günstigste Abtastebene rasch herauszufinden. Außerdem gestattet das A-Bild die für das Zustandekommen des B-Bildes maßgebenden Parameter wie Verstärkung, Schwellwert und Tiefenausgleich bereits vor dem Abtastvorgang optimal einzustellen. Andererseits gewährleistet das verzerrungsarme B-Bild immer eine ausreichende räumliche Orientierung, die die Zuordnung der Echos im A-Bild erleichtert. So ergänzen sich beide Methoden fortlaufend.

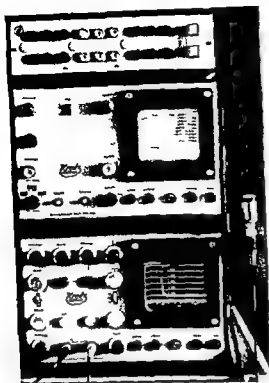


Abb 1 Ultraschall Schnittbildgerät mit Einrichtung für gleichzeitige A und B-Bildanzeigen (Fa. Kretztechnik Zipf Österreich)

Eine möglichst ruhige Lage des Kopfes bildet Voraussetzung für das Entstehen guter Ultraschalltomogramme. Der Patient wird daher im Liegen untersucht. Unruhige Säuglinge und Kleinkinder müssen sediert werden. Eine Rasur der Haare oder eine Wasererlaufstrecke sind nicht erforderlich. Das Untersuchungsgebiet wird mit einem der üblichen Elektroden Gelees präpariert.

Nach Lagerung des Kopfes wird die gewünschte Schnittebene eingestellt. Bei allen Patienten fertigen wir routinemässig ein Schnittbild in einer unteren horizontalen Ebene an, die zwischen 4 und 5 cm oberhalb des äußeren Gehörgangs liegt (untere Standardebene Abb 3). Bei Kindern werden zusätzliche Tomogramme in einer oberen Horizontalebene 6 bis 7 cm über dem äußeren Gehörgang (obere Standardebene Abb 3) sowie Vertikalschnitte vor und hinter dem Ohr angelegt. Dann stimmt man die individuelle Schallgeschwindigkeit des Untersuchungsobjekts auf die Prüfkopfbewegungen ab. Bei dickem Schädelsknochen muß eine gewisse Bilddehnung erfolgen, eine Stauchung dagegen bei Vorliegen eines besonders dünnen Schädels oder eines hochgradigen Hydrocephalus. Maßgebend für diese Korrektur sind die Verhältnisse im temporalen Bereich, da hier am ehesten mit einer Überlagerung der Echos von links und von rechts gerechnet werden muß.

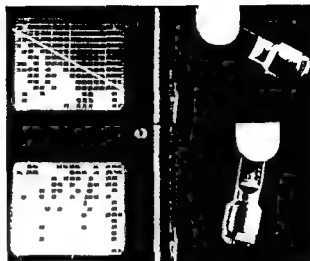


Abb. 2: Synchronisation von Irukkopfbewegungen und Zeitbasislilie bei der Compound Scanning Technik

Zu Beginn der Untersuchung wird unter Zuhilfenahme des Transmissionsverfahrens die theoretische Mittellinie des Schädels mit der Skalenmitte zur Deckung gebracht damit Abweichungen der von den Mittelstrukturen stammenden Echos nach rechts oder links sofort abgelesen werden können.

Die Ableitung in der unteren Standardebene zeigt in der Hauptsache Echos von den Mittellinienstrukturen und aus dem Trigonumbereich der Seitenventrikel wahrscheinlich vom Plexus chorioideus. Bei erweitertem Hirnkammersystem erkennt man darüberhinaus nicht selten die Seitenwände der Unterhörner. In der oberen Standardebene erhält man Echos von der Fissura interhemispherica bzw. der Falx und den Seitenventrikelwänden im Corpusbereich. Die verschiedenen Ventrikelschnitte bilden im wesentlichen die gleichen Strukturen ab. Hierdurch wird in manchen Fällen ein dreidimensionaler Eindruck des Ventrikelsystems möglich.

Die gesamte Untersuchung dauert bei Anfertigung von 6 bis 11 Schnittbildern maximal 30 bis 40 Minuten, während eine einzelne Aufnahme nach Einstellung aller Parameter innerhalb einer Minute herzustellen ist.

Bei der von uns verwendeten Prüffrequenz von 2 MHz sind der Methode durch die Dicke des Schädelsknochens Grenzen gesetzt. Übersteigt die Knochendicke 6 bis 7 mm, so lassen sich kaum noch Echos von Ventrikelwänden aufzeichnen. Das Verfahren ist daher vorwiegend für die Untersuchung von Kindern geeignet, allenfalls für Erwachsene mit dünnem Schädelsknochen. Mit niedrigeren Frequenzen, die eine größere Knochendicke zu durchdringen vermögen, konnten bisher keine zufriedenstellenden Resultate erzielt werden.

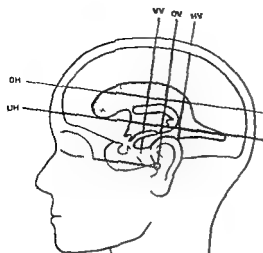


Abb 3 Schema der Schnittebenen für die Ultraschalltomographie UH = untere horizontale Standard Ebene OH = obere horizontale Standard Ebene OV = Ohrvertikale VV = vordere vertikale Schnitt Ebene HV = hintere vertikale Schnitt Ebene

Die folgenden Bildbeispiele aus einer Serie von mehr als 1 100 Ultraschalltomogrammen bei 200 Patienten sollen die Möglichkeiten des Verfahrens illustrieren

Besteht eine Massenverschiebung, so ist aus dem B-Bild häufig nicht nur das Ausmaß der Verlagerung abzulesen. Die unterschiedlich starke Verdrängung der einzelnen Mittelstrukturen läßt darüberhinaus Rückschlüsse auf den Ausgangspunkt der Massenverschiebung zu. So findet man beispielsweise bei temporalen raumfordernden Prozessen eine Mittellinienverlagerung, die nach frontal zu abnimmt, während bei frontalen Läsionen die Massenverschiebung im Stirnhirnbereich am stärksten ausgeprägt ist (Abb 4). Im Bereich des 3. Ventrikles oder des Corpus pineale braucht dabei nicht unbedingt eine Mittellinienverlagerung zu bestehen. Diese Befunde vermögen auch die Diskrepanzen zu erklären, die bei verschiedenen, kurz aufeinander folgenden Mittelechobestimmungen mit der A-Methode doch immer wieder zu beobachten sind. Die kombinierte A-Bild-Echographie gestattet also in manchen Fällen eine über den einfachen Seitenhinweis hinausgehende Lokalisation diagnose. Bei einer Vergleichsuntersuchung zwischen A- und B-Bild-Technik von 50 Kindern und Erwachsenen mit Großhirntumoren fand sich in 46 Fällen eine Übereinstimmung in der Richtung der Massenverschiebung. Das Ausmaß differierte aber zum Teil ganz erheblich, da im A-Bild meist nur die Position des Corpus pineale bestimmt wurde. In den verbleibenden 4 Fällen gelang es nicht, Echos von den Mittelstrukturen zweidimensional aufzuzeichnen, da der Schädelsknochen offenbar zu dick war. Im A-Bild konnte hier aber doch ein eindeutiges Mittelecho registriert werden, wenn der Prüfkopf direkt oberhalb des Ohres auf



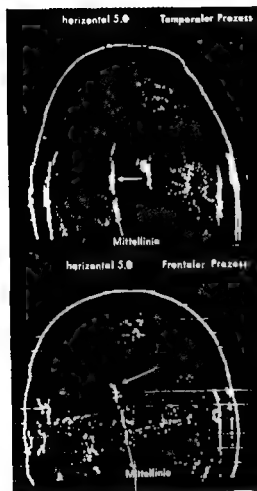


Abb. 4

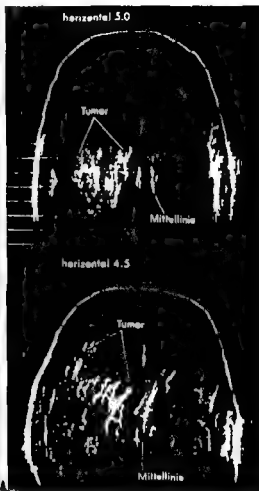


Abb. 5

Abb. 4 Unterschiedliches Verhalten der Mittellinie bei temporalen und frontalen raumfüllenden Prozessen sichtbar gemacht im Ultrachalltomogramm der unteren Standardebene

Abb. 5 Zwei Fälle von Astrozytomrezidiv im Ultraschallschnittbild

gesetzt und leicht nach oben gekippt wurde. Eine derartige Einstellung ist mit der tomographischen Technik nicht möglich, da hier das Ultraschallstrahlenbündel stets in der einmal gewählten Untersuchungsebene bleiben muß. Es ist also sicher auch von diesem Gesichtspunkt her wertvoll, beide Untersuchungstechniken zu kombinieren.

Von der eindimensionalen Echoencephalographie her wissen wir, daß bei Großhirngeschwülsten gelegentlich zusätzliche Reflexionen auftreten, die aus

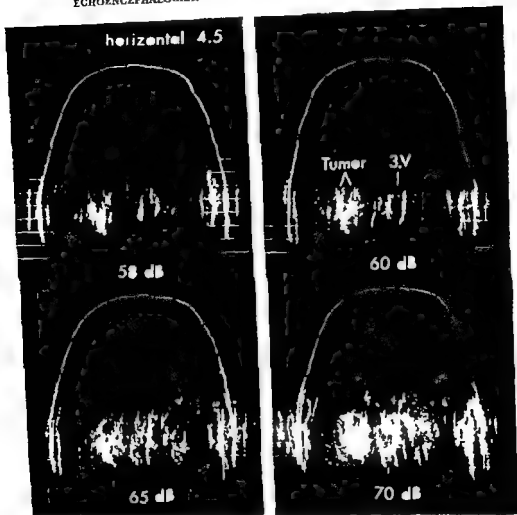


Abb 6 Einfluß der Impulsstärke auf die Abbildung eines Hirntumors. Bei zu hohen Impulsstärken ist keine klare Deutung des Ultraschallschnittbildes mehr möglich.

der Geschwulst selbst oder deren direkter Umgebung stammen. Für das Aufsuchen und Aufzeichnen derartiger abnormer Echo-Komplexe stellt die kombinierte A-B-Bild-Methode ein ideales Verfahren dar. Das Tumorgebiet wird im Schnittbild als eine Ansammlung von mehr oder weniger regelmäßig angeordneten hellen Punkten wiedergegeben, die nicht selten zu einem größeren hellen Fleck verschmelzen. Unter Berücksichtigung der gewählten Schnittebene kann eine gute räumliche Einordnung erfolgen und die Beziehung zu den Mittelstrukturen beurteilt werden. Auch hinsichtlich der Größe des Prozesses

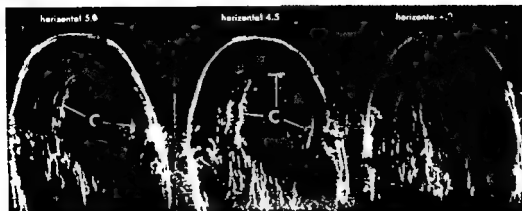


Abb 7 Darstellung eines großen zystischen Prozesses (C) im Ultraschallschnittbild. Es handelt sich um einen etwa  $170 \text{ cm}^3$  großen Abszeß im linken Stirnhirn. Besonders deutlich ist hier die nach vorne stark zunehmende Mittellinienverlagerung (rechtes Bild) durch die frontale Raumforderung.

sind Rückschlüsse möglich, nicht jedoch in Bezug auf die histologische Feinstruktur. Die beiden in Abb 5 gezeigten Fälle waren Gliomrezidive bei Erwachsenen, die anlässlich einer ambulanten Routinekontrolle entdeckt wurden. Die Tumoren heben sich durch die abnormen Echoansammlungen vom echofreien Untergrund deutlich ab.

Bei der Aufzeichnung von Tumorechos muß der Impulsstärke größte Beachtung geschenkt werden. Die Echogrammserie in Abb 6 zeigt 4 Abtastbilder eines 10-jährigen Mädchens alle in der gleichen Schnittebene. Die Patientin kam mit Zeichen des gesteigerten intrakraniellen Druckes und dem Verdacht auf das Vorliegen eines raumfordernden Prozesses im Bereich der hinteren Schädelsgrube bei uns zur Aufnahme. Die Ultraschalltomogramme zeigten jedoch nur normale Echos in der Tiefe des rechten Schläfenlappens. Bei der Operation konnte ein hühnereigroßes Plexuspapillom aus dem rechten Temporalhorn entfernt werden. Es ist bemerkenswert, wie unter dem Einfluß steigender Impulsstärke die Ausdehnung des Echo-Komplexes aus dem Tumorgebiet immer größer wird. Die Bilder sind entschieden einfacher zu beurteilen, wenn man die Untersuchung mit einer möglichst niedrigen Impulsstärke beginnt und dann langsam steigert.

Ob kleinere Zysten bis zu einem Durchmesser von etwa 1 cm in einem soliden Tumor durch eine entsprechende Echoausparung zu erkennen sind, läßt sich nach den bisherigen Erfahrungen noch nicht mit Sicherheit sagen. Größere Zysten sind aber klar als echo-freie Zonen, die von einem mehr oder weniger geschlossenen Ring von Echos umgeben sind, zu identifizieren. Die Bildserie in Abb 7 stammt von einem 3-jährigen Jungen mit einem etwa  $120 \text{ cm}^3$  großen

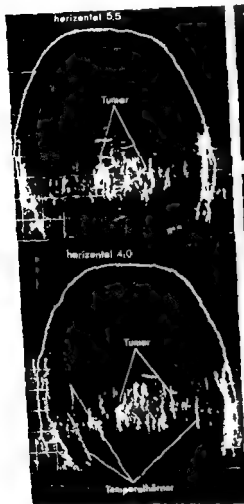


Abb 8



Abb 9

Abb 8 Aufzeichnung eines Tumors im 3 Ventrikel im Ultraschallschnittbild. Auf dem unteren Tomogramm erkennt man neben dem großen Tumorecho-Komplex feine Linien, die von den erweiterten Unterhörnern stammen.

Abb 9 A- und B-Bilddarstellung einer Ventrikelvergrößerung. Ableitung in der oberen Standardebene. Die Seitenventrikelwände, die weitgehend parallel zur Mittellinie verlaufen, lassen sich im B-Bild über eine Strecke von mehreren Zentimetern aufzeichnen.

Abzess im linken Stirnhirn. Auf den Schnittbildern ist ganz deutlich eine große Zyste in den linken vorderen Hirnabschnitten zu sehen. Außerdem erkennt man auf diesen Bildern die nach vorne zu stark zunehmende Mittellinienvverlagerung, besonders klar.

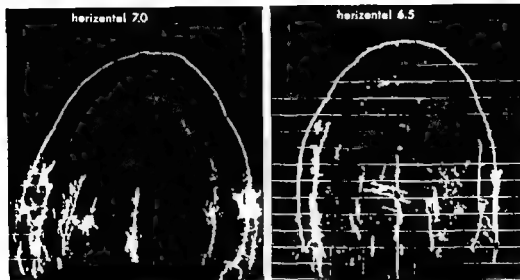


Abb 10 Sichtbarmachung des Ventrikulkatheters innerhalb der erweiterten Hirnkammern bei Kindern nach Shuntoperation. Der Pfeil weist jeweils auf den Katheter bzw. die Katheterspitze hin.

Auch bei Prozessen im 3. Ventrikel oder dessen unmittelbarer Nachbarschaft hat sich die Scanning-Methode bewährt. Abb. 11 zeigt Ultraschalltomogramme eines 3-jährigen Jungen in zwei verschiedenen Horizontalebene mit einer Formation abnormer Echos im Mittellinienbereich, der im Szintigramm eine umschriebene Impulsanreicherung an gleicher Stelle entsprach. Bei der Operation durch den hinteren Balken konnte ein apfelgroßes Pineoblastom entfernt werden. Das untere Bild zeigt bereits Echos, die von den Außenwänden der erweiterten Temporalhörner stammen.

Wir kommen damit zu einer der wichtigsten Indikationen der kombinierten A-Bild-Echographie: dem kindlichen Hydrocephalus. Entscheidend für die Diagnose aus dem Ultraschallbild ist hier das Tomogramm der oberen Standardebene. Zunächst sucht man im A-Bild in der Temporo-Parietalregion die charakteristischen Echos auf, um dann in dieser Höhe den Prüfkopf um den Schädel herumzuführen. Wie aus Abb. 9 zu ersehen ist, vermag das zweidimensionale Bild einen wesentlich besseren Eindruck der Form und Größe des Ventrikelsystems im Verhältnis zum gesamten Schädel zu vermitteln als das relativ abstrakte A-Bild. Auch Asymmetrien sind leicht zu erkennen. In der unteren Standardebene können zusätzlich die Wände des 3. Ventrikels, das Septum pellucidum und die Wände der Unterhörner aufgezeichnet werden. Mit der Methode gelang auch erstmals die einwandfreie Darstellung einer

großen Septum pellucidum Zyste Nach unseren bisherigen Erfahrungen an 67 Kindern mit Hydrocephalus können die Ventrikelkonturen im ersten Lebensjahrzehnt fast immer im II Bild aufgezeichnet werden Der älteste Patient der Serie mit klarer Ventrikeldarstellung im Ultraschalltomogramm war 15 Jahre Die Schnittbilder dieses Patienten zeigten die Konturen der massiv erweiterten seitlichen Hirnkammern sogar über eine außergewöhnlich lange Strecke

Bei Kindern mit Shunt Operationen bei Hydrocephalus eröffnet die Schnittbildchographie eine weitere diagnostische Möglichkeit Hier kann die Lage des Ventrikelkatheters in Beziehung zum Ventrikelsystem beurteilt werden Abb 10 oben zeigt Echos einer Ventrikelkatheterspitze die nur wenig in den rechten Seitenventrikel hineinragt und deutlich verdickt ist Dieser Befund wurde durch eingewachsenes Plexusgewebe hervorgerufen Beim rechts in Abb 10 abgebildeten Fall reicht der Ventrikelkatheter bis in den linken Seitenventrikel hin über Bemerkenswert ist daß sowohl Vorder als auch Rückwand des nur 2,5 mm dicken Silikongummischlauches getrennte Reflexionen ergeben

Die Kombination von gleichzeitiger ein und zweidimensionaler Echoencephalographie in der Diagnostik intrakranieller Prozesse scheint eine wertvolle Ergänzung der bisherigen Beschallungstechniken darzustellen Die Methode hat zwar keine so umfassenden Indikationen wie die einfache A Bild Technik — sie ist zum Beispiel nicht für die Untersuchung unruhiger Schädel Hirnverletzter geeignet — sie erhöht aber zweifellos Objektivität und Informationswert echoencephalographischer Befunde bei zahlreichen Fällen beträchtlich

Die besten Ergebnisse wurden bisher bei Säuglingen und Kleinkindern mit noch relativ dünner Schädelkalotte erzielt, wobei es in einzelnen Fällen sogar möglich war Strukturen abzubilden deren Sichtbarmachung mit den Kontrastmittelmethode kaum möglich ist Andererseits wird aber die Ultrachalltomographie niemals Bilder von der Exaktheit eines Encephalogramms liefern können Die Echoencephalographie ist daher nicht als Konkurrenz zu den neuroradiologischen Untersuchungsverfahren, sondern als wichtiges Hilfsmittel bei der Entscheidung über die Wahl der weiteren diagnostischen und therapeutischen Schritte anzusehen Bei Erwachsenen ist die Anwendung des B Bild Verfahrens wegen der Dicke der Schädelkalotte problematisch Bei Kindern stellt die Ultrachalltomographie als Screening Test und in der Langzeitbeobachtung nach Tumor oder Shunt Operation eine wesentliche Bereicherung der ambulant durchführbaren technischen Hilfsuntersuchungen dar

### ZUSAMMENFASSUNG

Es wird über eine Ultraschalluntersuchungsmethode am Schädel unter gleichzeitiger Anwendung des A und B Bild Verfahrens berichtet Hierdurch kann das Problem die zahlreichen bei der eindimensionalen Echoencephalographie auftretenden Reflexionen den ent

sprechenden intrakraniellen Strukturen zuzuordnen besser gelöst werden als mit den bisher bekannten Methoden. Das II Bild entsteht auf einem Speicherschirm nach der Compound Scanning Technik. Als besonders wertvoll hat sich die kombinierte A-B Bild Methode beim kindlichen Hydrocephalus sowie bei supratentoriellen Tumoren, Zysten und Abszessen bei Kindern und Jugendlichen erwiesen, während dem Verfahren bei Erwachsenen durch die Dicke des Schädelsknochens Grenzen gesetzt sind.

## SUMMARY

An ultrasonic method of skull examination with simultaneous A and B scanning is described. The origin of the numerous echoes in the A scope can be more easily determined than by the common methods. The compound scanning technique is used. The ultrasonotomogram is displayed on a memory tube. The combined A-B method has proved of value in infantile hydrocephalus, supratentorial tumours, cysts and abscesses in children and juveniles. The thickness of the cranium rather limits the method in adults.

## RÉSUMÉ

L'auteur présente une méthode d'examen du crâne par les ultrasons utilisant simultanément les techniques d'échographie A et B. Cette méthode permet mieux que les méthodes utilisées jusqu'à maintenant de résoudre le problème qui consiste à rattacher aux structures intra-crâniennes correspondantes les nombreuses réflexions qui se produisent dans l'écho-céphalographie unidimensionnelle. L'image B se forme sur un écran à rémanence selon la technique de compound scanning. La méthode combinée d'échographie A et B s'est montrée particulièrement utile dans l'hydrocéphalie de l'enfant ainsi que dans les tumeurs supratentorielles, les kystes et les abcès de l'enfant et du jeune, alors que les possibilités de cette méthode sont limitées chez l'adulte par l'épaisseur de la voûte du crâne.

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## TRANSORBITALE ECHOENCEPHALOGRAPHIE

von

H. R. MÜLLER und W. OSTHEIM

Die durch Form und physikalische Eigenschaften der Kalotte beschränkten Möglichkeiten der Echoencephalographie am geschlossenen Schädel (WHITE et coll. 1967, DE VLIÉGER 1969) veranlassten uns, als weiteren sonographischen Zugang zum Endokranium den transorbitalen Weg zu erproben.

Bei orientierenden Lotungen an gesunden Versuchspersonen und neurochirurgischen Patienten zeigte sich dabei, dass das Schallbündel einer Sonde von 2 MHz und 1,5 cm Schwingerdurchmesser bei den in der Diagnostik üblichen Impulsstärken den Bulbus oculi, die retrobulbären Weichteile und die knöchernen Orbitahüllen zu durchdringen vermag und dass sich bei geeignetem Einfallswinkel von der Tabula interna des Os parietale ein energiereiches Echo auffangen lässt (MÜLLER 1969, 1970).

Dies schien um so erstaunlicher, als bei den erprobten Lotungsrichtungen das knöcherne Orbitaskelett in einem weiten Bereich unter einem Einfallswinkel beschallt wurde, der den auf 30° zu veranschlagenden kritischen Wert (WHITE 1971) unterschritt und deshalb eine Totalreflexion erwarten liess.

Bevor wir die Methode deshalb klinisch anwendeten, schien es uns angezeigt, die Auswirkungen des Orbitaskelettes auf das Schallbündel experimentell zu untersuchen. Hierzu bedienten wir uns folgender Vorrichtung:

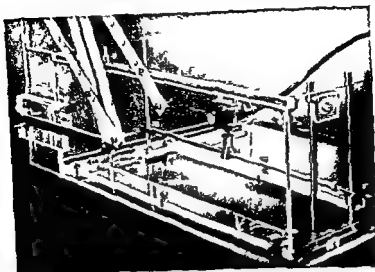


Abb 1 Gerät zur Bestimmung der Schallfeldausseengrenzen SK = Schallkopf M = Magnet der von der Aussenseite der Frontwand die im innern des Wassertanks befindliche Stahlkugel k. festhält. P = Pantograph. S = Schreibstift

In einem aus Plexiglas bestehenden Wassertank (Abb 1) wurde senkrecht auf eine Seitenwand zuführend eine Bodenschuene angebracht auf der sich die an einem Stativ befestigte Sonde eines Impulsehogerates in wählbare Abstände zu dieser Seitenwand bringen liess Mittels eines Kugelgelenkes konnte die Ultraschallsonde ausserdem während des Impulsehobetriebes unter Kontrolle am Bildschirm genau senkrecht auf die Frontwand des Wassertanks justiert werden Auf der Aussenseite der zu beschallenden Wand wurde ein mit einem Pantographen verbundener Stabmagnet montiert der sich durch manuelle Führung des Pantographen in beliebige Positionen zur Schallachse bringen liess Dieser Magnet hielt auf der Innenseite der Wand eine Stahlkugel von 3 mm Durchmesser fest die die Positionsänderung des Pantographen, kraft der Auswirkung des magnetischen Feldes, mitmachte

Mit diesem Gerät liessen sich die Ausseengrenzen des effektiven Schallfeldes (WHITE 1971) bestimmen indem die Kugel systematisch in der zum Schallkopf senkrechten Führungsebene von allen Seiten der Schallachse genahert wurde Trat dabei ein dem Wandecho vorgelagertes und damit dem kugelförmigen Reflektor zuzuordnendes Signal auf so wurde mit dem Schreiber des Pantographen eine Marke angebracht Die Verbindung aller Marken durch eine Linie ergab die Ausseengrenze des bei einer gegebenen Impulsstärke und Verstärker Einstellung wahrnehmbar reflektierenden Schallfeldes



Abb. 2 Schallfeldausseengrenzen bei transorbitaler Lotung in verschiedenen Richtungen. Schallkopf 2 MHz, 15 mm Schwingerdurchmesser. Abtastung des Schallfeldes in einer der Parietalregion entsprechenden Tiefe.

Wurde nun der Schallkopf in einen Abstand zur reflektierenden Wand des Wassertanks gebracht, welche dem Abstand der Bulbusvorderfläche zur parieto-occipitalen Konvexität ungefähr gleich war und interponierte man zwischen Schallkopf und Wand ein Orbitaskelett in der Weise, dass die Einfallswinkel des Schallbündels denjenigen bei den zu erprobenden Lotungsrichtungen entsprachen, dann konnte die Auswirkung dieses akustischen Hindernisses auf das Schallfeld untersucht werden.

Auf Grund der mit dieser Methode gewonnenen Ergebnisse, die nach ihrer Vervollständigung in extenso publiziert werden sollen, ist es offensichtlich, dass das Schallbündel zwar eine nicht unerhebliche Distorsion und ungleichmässige Abschwächung erfährt, dass aber eine Achse nicht nennenswert abgelenkt wird (Abb. 2).

Während diese Untersuchungen noch immer weitergeführt werden, sind wir deshalb inzwischen zu der klinischen Prüfung der transorbitalen Echoencephalographie zurückgekehrt und haben eine Reihe von Befunden zusammengetragen, welche die Methode als recht aussichtsreich erscheinen lassen.

Die bis jetzt erprobten Techniken und Anwendungen sind die folgenden:

**Transorbitale A-Scope Echoencephalographie zur Feststellung chronisch subduraler Hamatome.** Bringt man den Schallkopf unter Verwendung von Methocel in Kontakt mit dem geschlossenen Lid, so kann bei Lotung in sagittalem Strahlengang in einem zur Deutschen Horizontalen etwa 20–40° betragenden Höhenwinkel ein Echo von der Tabula interna der Parietalregion



Abb 3 Transorbitale A-Scope-Sonogramme eines chronisch subduralen Hamatoms rechts a) Seite des Hamatoms Die Lotung erfolgte in einem Hohenwinkel von 30° zur Deutschen Horizontale Die Seitenwinkel sind auf den einzelnen Sonogrammen angegeben II = Hamatoecho B = Knochenecho b) Gesunde Seite Lotung in jeweils symmetrischer Einstellung zu derjenigen auf der Hamatomseite

aufgefangen werden. Schwenkt man das Schallbündel bei unveränderter Lotungsposition nach außen, dann pflegt dieses Echo dem Nullpunkt zunächst unter Amplitudenabnahme etwas entgegenzuwandern, um bei den meisten Schädelformen bei einem Seitenwinkel zwischen 15° und 30° zu verschwinden (Abb 3 b).

Demgegenüber liess sich bei chronisch subduralen Hamatomen ein wohl der Membran zuzuordnendes stabiles Endecho registrieren, welches sich bei Lateralschwenkung des Schallkopfes bis zu einem Seitenwinkel von 30° beträchtlich auf den Nullpunkt zu verlagert und in der Amplitude meist kaum reduziert wird (Abb 3 a). Die Erklärung für diese Beobachtung findet sich in Abb 4 schematisch erläutert. Während sich die Kalotte entsprechend einem

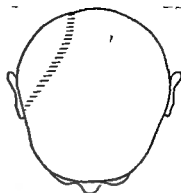


Abb 4 Schematische Darstellung der den Sonogrammen von Abb 3 zugrunde liegenden Reflexionsverhältnisse  
Schraffiert dargestellt Hamatommembran

Hohlspiegel verhält und deshalb von einem kritischen Seitenwinkel an überhaupt keine Energie mehr auf den Schallkopf zurückwirft, scheinen Oberfläche und akustische Innenstruktur der Hamatommembran genügend unregelmässig zu sein, um auch bei starker vom Lot abweichenden Einfallswinkeln noch Energie in der Lotungsrichtung zu reflektieren.

Bei den bisher transorbital geloteten 7 chronisch subduralen Hämatomen liess sich das beschriebene Phänomen nur in einem Fall nicht nachweisen, ohne dass dafür eine befriedigende Erklärung gegeben werden konnte.

*Transorbitale Sektor B-Scan Echoencephalographie zur Darstellung parietaler Tumore und chronisch subduraler Hämatome* Eine weitere mögliche Anwendung des transorbitalen Lotungsweges, welche die klinische Erprobung verdient, stellt das B-Scan Verfahren dar. Am meisten Aussichten, signifikante Resultate zu geben, scheint dabei die Sektor Technik aufzuweisen, sei es als Contact Scanning mit direktem Aufsetzen der Sonde auf das geschlossene Lid oder sei es als Waterpath Scanning unter Verwendung einer wassergefüllten Taucherbrille. Die bisherigen Untersuchungen mit diesem Verfahren erfolgten mit einem manuell geführten Scanner (Laminograph Picker). Obschon sie in einzelnen Fällen recht eindruckliche Resultate ergaben (MÜLLER 1970), bieten sich in der korrekten Winklereinstellung, der gleichmässigen Bewegung des Schallkopfes und der Beibehaltung des akustischen Kontaktes ganz beträchtliche Schwierigkeiten. Durch die Verwendung eines in Konstruktion befindlichen motorbetriebenen Scanners hoffen wir, denselben wirksam zu begegnen.

Dieses Gerät wird es gestatten, zwei synchron bewegte, aber wechselweise elektroakustisch betriebene Schallköpfe in genau definierten Schnittebenen zu führen und die im Bereiche beider Schädelhälften erhobenen Befunde laufend auf einem und demselben Bildschirm zu vergleichen.

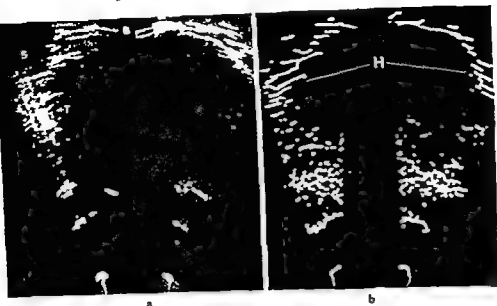


Abb 5 Transorbitale Sector B-Scans a) Meningeom parietal rechts T = Tumorecho S = akustischer Schatten B = Knochenecho b) Doppelseitiges chronisch subdurales Hamatom H = Hamatomecho

Die transorbitale Sector B-Scan Methode verspricht besonders Aufschluss über parietale Tumoren (Abb 5 a) und konnte gerade hier zu einer wertvollen Ergänzungsuntersuchung zu den neuroradiologischen Techniken werden. Im Erprobungsstadium hat sie sich ausserdem bei chronisch subduralen Hämatomen bewährt (Abb 5 b).

Abschliessend lässt sich auf Grund unserer vorläufigen Erfahrungen feststellen, dass der transorbitale Lotungsweg sowohl für die A-Scope- als auch für die B-Scan Technik Aussichten auf eine Erweiterung der echoencephalographischen Information bietet. Wahrscheinlich ist das erfassbare Gebiet aber weitgehend auf die Parietalregion beschränkt.

## ZUSAMMENFASSUNG

Bei Abtastung des Schallfeldes mittels eines kugelförmigen Reflektors liess sich experimentell zeigen, dass das Schallbündel einer 15 mm breiten Sonde von 2 MHz, welches durch die Orbita nach der Parietalregion gerichtet wird, durch die knöchernen Strukturen zwar eine unregelmässige Abschwächung und Distorsion erleidet, dass aber die Richtung der Schallachse nicht wesentlich abgelenkt wird. Die transorbitale Echoencephalographie wurde an normalen Probanden und an einem neurochirurgischen Krankengut erprobt. Chronisch subdurale Hämatome und parietale Tumoren liessen sich damit nachweisen.

## SUMMARY

The plotting of the ultrasonic field with a spherical reflector demonstrated that a sonic beam of 2 MHz emitted by a 15 mm probe and directed transorbitally towards the parietal area suffers some irregular attenuation and distortion but that its direction remains essentially unaltered. Extensive tests of transorbital echo encephalography were undertaken in normal controls and a material of neurosurgical cases. Good results were obtained in chronic subdural haematomas and parietal tumours.

## RÉSUMÉ

L'exploration du champ ultra sonore au moyen d'un réflecteur sphérique a montré expérimentalement que le faisceau ultra sonore d'une sonde de 2 MHz large de 15 mm, dirigé à travers l'orbite vers la région pariétale subit du fait des structures osseuses un affaiblissement irrégulier et une distorsion mais la direction de l'axe du faisceau n'est pas notablement déviée. L'écho-encéphalographie transorbitaire a été expérimentée sur des sujets normaux et sur des malades neuro chirurgicaux. Elle permet de détecter les hématomes sous duraux chroniques et les tumeurs pariétales.

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## EXPERIENCE WITH INTRAOPERATIVE ECHOENCEPHALOGRAPHY IN CEREBRAL MASS LESIONS

by

R. L. SCHLAGENHAUFF and F. E. GLASALER

In 1951 FRENCH and his co-workers suggested the diagnostic application of ultrasound during surgery. Since then several investigators have demonstrated different acoustic impedances for normal and neoplastic brain tissue thus establishing the usefulness of ultrasound for localization of cerebral mass lesions (2, 5-14). Differences in impedance due to abrupt change in tissue density, produce prominent echoes on the oscilloscope. The present investigation was undertaken to confirm the usefulness of intraoperative ultrasonic exploration.

### Method

The ultrasonic instrument used is an Ekoline 20 (Smith Kline Precision Co.) and it operates on a frequency of 2.25 Mc/sec representing linear A mode. The transducer measures 2.6 cm in diameter and the diameter of the sound beam at the point of transmission is 1.3 cm. We also used a smaller probe with a diameter of 0.5 cm and a sound beam of 0.3 cm. Although the small diameter of the probe allowed examinations through a regular burr hole opening, we found it to be less sensitive and therefore ineffective for our purpose. A high frequency is



Table

*Pathologic diagnosis of investigated lesions*

Diagnosis	Number of cases
Gliomatous tumor	13
Meningioma	7
Miscellaneous tumors	8
Intracerebral hematoma	4
Abscess	1
Infarction (including cystic)	3
Aneurysm	1
Subdural hematoma	3
	40

required to produce sharper interphases whereas a better tissue penetration is obtained with lower frequencies (6). We, therefore, knowingly forego the latter for the benefit of a better interphase by using 2.25 Mc/sec.

Echoencephalographic explorations during surgery referred to as intraoperative echoencephalography, were carried out after elevation of the bone flap either transdurally or, on a few occasions directly on the cerebral cortex (5). The test is relatively simple and requires about 10 minutes time. Bacterial contamination or damage to the transducer by autoclaving is avoided by gas sterilization. Both the membrane of the transducer and the dura are moistened with sterile saline and the transducer is lightly applied to the dura of the brain surface. During gentle rocking movements the ultrasonic reflections are observed, and photographed on polaroid film. The operative field is systematically probed in this manner at multiple sites and the observations are compared with the operative findings.

## Results

Intraoperative echoencephalography was performed in a selective group of 40 patients. The pathologic diagnoses of the various supratentorial space occupying lesions are listed in the Table. Five representative cases are illustrated.

**Case 1.** A 57 year-old man complained of headaches and visual difficulty and examination substantiated a right homonymous hemianopsia. Echogram disclosed a 2 mm shift of the midline structures to the right and a  $^{201}$ Hg brain scan showed a fairly well circumscribed moderately intense abnormal uptake in the left occipital lobe. On the angiogram a collection of abnormal vessels in the same area was evident. Through a craniotomy a large portion of

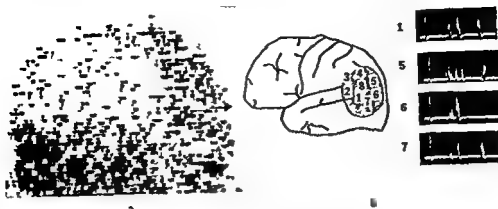


Fig 1 a) Left lateral brain scan with  $^{197}\text{Hg}$  demonstrates an abnormal uptake in the occipital lobe (Posterior  $\rightarrow$ ) b) The lesion is outlined on the sketch and the numbers refer to the points of echoencephalographic probings. Prominent echo reflections are seen at a depth of 4 to 5 cm at the pertinent sites. (Each marker of the baseline represents 1 cm)

the tumor mass together with the left occipital pole was removed. The histologic diagnosis was glioblastoma multiforme. At intraoperative echoencephalography prominent echo reflections were seen at a depth of 4 to 5 cm at the tumor site (Fig 1).

**Case 2** This 77 year old man suffered from progressive mental deterioration and nocturnal seizures for several years. Examination confirmed the severe dementia and disorientation and there were marked grasping, groping and sucking reflexes. Brain scan showed increased uptake in the left frontal pole and carotid angiography revealed the presence of a left subfrontal tumor extending across the midline. Through a bifrontal craniotomy a large falx meningioma was removed and the echo reflections obtained correlated well with the operative findings (Fig 2).

**Case 3** A 59 year old man was admitted for complaints of headaches and roentgen examination of the chest revealed a mass, presumably a bronchogenic tumor. Carotid angiography demonstrated abnormal vessels in the right parietal area. This was consistent with an abnormal uptake on the brain scan. At craniotomy a large cerebral metastasis was removed from the right parietal area and the histologic diagnosis was bronchogenic carcinoma. Before the cortical incision was made ultrasonic signals were prominent at a depth of 4.5 cm over the tumor mass (Fig 3).

**Case 4** Two weeks before admission this 41 year-old man had an episode of chills and complained of tiredness. He was admitted for possible alcoholism and convulsions. The neurologic examination was normal, except for focal right sided seizures. Later on he became lethargic and confused. A brain scan showed a positive uptake in the left frontal area and at carotid angiography an avascular mass in the left frontal lobe was found. A left frontal craniotomy was carried out. At intraoperative echoencephalography significant echo signals were found over the suggested area and an intracerebral abscess was removed (Fig 4).



a

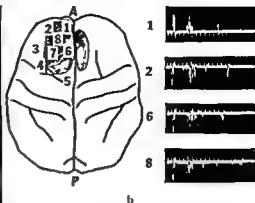


Fig 2 a) Ap view of left carotid angiography reveals a highly vascularized tumor in the left frontal area consistent with a meningioma b) Marked echoes at a depth of 3.8 cm correlating well with the operative findings



a

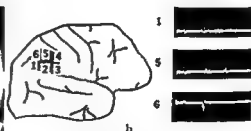
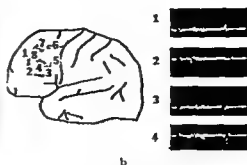


Fig 3 Lateral view of right carotid angiography reveals abnormal vessels in the right parietal area Metastatic bronchogenic carcinoma b) Significant ultrasonic signals are seen at a depth of 4.5 cm over the metastatic lesion



a

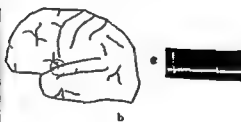


b

Fig 4 a) An avascular mass is demonstrated in the frontal area at lateral carotid angiography b) Good echo signals are found over the suggested site correlating with an intracerebral abscess



a



b

Fig 5 a) Ap view of left carotid angiography reveals a subdural hematoma b) Echoencephalography through an enlarged burr hole corresponds to the thickness of the subdural hematoma before evacuation

*Case 5.* A 38 year-old woman gave a history of a recent fall in which she sustained a scalp laceration and a brief loss of consciousness. Examination revealed disorientation, a left hemiparesis and a third nerve palsy. Echoencephalography revealed a 6 mm shift of the midline structures to the right. Bilateral carotid angiography revealed a subdural hematoma on the left side. Echoencephalography through the burr hole opening substantiated the thickness of the subdural hematoma before evacuation (Fig. 5).

### Discussion

The clinical value of ultrasonic determination of cerebral midline structures and measurements of the third and lateral ventricles have now been well established (1, 3, 7, 9, 10, 13). The accuracy of this method in these examinations, however, has varied in different reports (11, 12, 15). Refined radiologic examinations produce quite accurate preoperative knowledge of the site and extent of intracranial mass lesions. Intraoperative ultrasonic evaluation can provide additional information as to the depth and extent of an intracranial tumor and aid in the localization of intracerebral hemorrhage or abscess. An aspiration needle may be inserted at an established point where the hematoma appears nearest to the surface and single or multi loculated abscesses may also be recognized by this method. This advantage seems especially valuable in the presence of marked increased intracranial pressure. In operations on the dominant hemisphere, ultrasonic mapping may help in selecting the cortical incision closest to the underlying tumor and thus preserves valuable brain tissue.

It is beyond the capability of this method to distinguish echoes produced by tumors from those produced by hematomas and other lesions or to establish a differential diagnosis of tumors. Although the more malignant astrocytomas produce more prominent echoes the reliability of this technique in low grade astrocytoma remains to be confirmed by a larger series. We presume, however, that these tumors have a different acoustic impedance and even with low grade malignancy they produce definite echoes. We were also unable to demonstrate the larger baseline undulations in cases of hematomas described by WATKINS & UEMATSU (1966). Cerebral edema, associated with tumor, abscess, or hemorrhage, has not been a significant impediment to the ultrasonic localization.

Although the transdural echoencephalographic technique minimizes the possibility of manipulative trauma or contact infection, cortical application of the transducer has not resulted in subpial hemorrhages. No significant wound infections attributable to the ultrasonic probing have been observed in this series.

### Conclusions

Our findings confirm those of previous authors. Intraoperative echoencephalography is useful in selection of the appropriate operative site on the dominant hemisphere and as an aid in the diagnosis of the following conditions: (1)

primary brain tumor, (2) metastatic brain tumor, (3) intracerebral hematoma, and (4) intracerebral abscess. Transdural echoencephalography may also prove helpful in the search for foreign bodies, including embedded bone fragments (JACKSON *et coll* 1965), and in the guidance of a biopsy needle. Further refinement of the method and experience with a lower frequency may produce additional information and widen the range of its application.

### SUMMARY

A selective group of 40 patients with various supratentorial space-occupying lesions was examined by transdural or cortical echoencephalography during craniotomies or trephinations. The examination proved helpful in the selection of the cortical incision site, especially in the dominant cerebral hemisphere and in outlining the depth and extent of various expanding lesions. The accuracy of the method was found sufficient and may improve with further refinement and experience.

### ZUSAMMENFASSUNG

Eine ausgewählte Gruppe von 40 Patienten mit verschiedenen supratentoriellen raum einengenden Schäden wurden im Zusammenhang mit einer Craniotomie oder Trepanation mit transduraler oder corticaler Echoencephalographie untersucht. Die Untersuchung wurde bei der Wahl der corticalen Incision besonders der überdeckenden cerebralen Hemisphäre und bei der Abgrenzung der Tiefe und Grösse der verschiedenen expandierenden Schäden, als nützlich befunden. Die Genauigkeit der Methode wurde als hinreichend beurteilt und mag durch weitere Verfeinerung und Erfahrung zu verbessern sein.

### RÉSUMÉ

Un groupe sélectionné de 40 malades atteints de différentes tumeurs sus-tentorielles a été examiné par écho-encephalographie transdurale ou corticale au cours de craniotomies ou de trepanations. Cet examen s'est montré utile pour choisir le lieu de l'incision corticale en particulier sur l'hémisphère dominant et pour délimiter la profondeur et l'étendue de différentes lésions expansives. La précision de cette méthode s'est montrée suffisante et peut s'améliorer avec des perfectionnements ultérieurs et avec l'expérience.

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## EXPERIMENTAL ULTRASONIC INJURY AND SAFETY LIMITS IN ITS USE

by

K. J. W. TAYLOR and J. B. POND

Ultrasound is now widely used in physiotherapy for the treatment of Meniere's disease and more recently, for diagnosis. In the 1940's it was employed in the treatment of malignancy, usually with good results but with occasional disaster when its application was followed by general dissemination. However much work on the fundamental way in which ultrasound interacts with solid tissue remains to be done: this is a complex biophysical problem in which calibration methods are non-uniform and hence results from different laboratories are difficult to reproduce.

The effects of ultrasound on any medium are either thermal or mechanical. The continuous beam used in much past experimental work has inflicted purely thermal damage. This bulk heating due to absorption of acoustic energy rises with increasing frequency. However, heating is of no significance in diagnosis when the small amount generated in a short pulse is dissipated in the relatively long interval between adjacent pulses.

Of the mechanical effects of ultrasound on biologic material only cavitation has been considered at length. Again this is of no relevance to its diagnostic use for it is most unlikely that cavitation would be supported by very short pulses of this intensity and frequency in a viscous biologic medium. Other mechanical forces such as oscillatory disturbances occur and a number of time averaged for



ces are exerted as a result of these primary cyclic forces acting on non linearities and inhomogeneities in the medium. These time averaged forces are considered in further publications. The results reported here are believed to be due to oscillatory disturbances. This term refers to the cyclic variations of temperature, pressure, velocity, displacement and acceleration which oscillate around their ambient value but give rise to no time averaged displacement. These should be considered since they are of large magnitude. For a typical diagnostic regime using a frequency of 1 MHz and a peak intensity of  $50 \text{ W cm}^{-2}$ , then the oscillatory variation in pressure will be some 12 atmospheres, the velocity  $82 \text{ cm s}^{-1}$ , and the oscillatory variation in acceleration 520 000 gravities. Since cells and organelles are exposed to such forces it is indeed surprising that injury does not occur. Nevertheless it is important that not only immediate injury be considered, the possibility of long term accumulation with genetic aberrations must also be considered.

In this work a frequency range of 0.5 to 6 MHz was used and a peak intensity of 25 or  $50 \text{ W cm}^{-2}$  which is the same range as that employed by diagnostic devices. Delivery of energy was pulsed 10 ms on and 100 ms off, both to avoid thermal effects and to simulate diagnostic usage. However the pulses used were much longer than those for diagnosis so the energy delivered could be increased sufficiently to produce damage and hence identify any thresholds. When oscillatory movements are considered the size of the irradiated structure complex may be of importance. In a cell suspension individual cells will be subject only to point displacement since they are free to move in relation to each other. In intact tissue when a number of cells are joined firmly together by complex interdigitations a structure complex of many cells is formed which is no longer small in relation to the wavelength and hence may be prone to greater disruption than a single cell subject to only point displacement. This may invalidate much of the past toxicity work in which cell suspensions have been utilised as an experimental model since clinically ultrasound is used on intact tissue composed of large structure complexes.

On the contrary spinal cord presents an excellent experimental model since irradiation with ultrasound of relatively low frequencies first produces functional damage indicated by paraplegia, while further exposure results in gross haemorrhage into the cord. A typical haemorrhage is illustrated in Fig. 1 and is seen to be predominantly in the grey matter. The occurrence of haemorrhage was found to be a precisely consistent and immediately visible criterion of injury sustained. This was therefore accepted as the end point of ultrasonic damage for the accurate comparison of the effects of treatment while varying each parameter of treatment. It is possible that this vascular injury explains the occasional occurrence of metastases referred to previously when tumours were irradiated with



Fig 1 I/S Rat spinal cord with gross haemorrhage into the grey matter following irradiation with ultrasound at frequency 1 MHz peak intensity  $25 \text{ W cm}^{-2}$  (pulsed 1:10) for four min

ultrasound of 0.8 or 1.0 MHz frequency these vascular defects would easily allow malignant emboli to enter the circulation aided by microstreaming and radiation pressure

The effect of varying frequency alone was investigated initially with constant peak intensity of  $50 \text{ W cm}^{-2}$  pulsed 1:10 delivering a time averaged intensity of  $5.0 \text{ W cm}^{-2}$ . The exposed spinal cord was irradiated at frequencies of 0.5, 1.2, 3.5, 4.2 and 4.9 MHz the total exposure time for production of gross haemorrhage being determined for each frequency. Since the pulsing regime (1:10) reduced the actual component of irradiation time received by a factor of 10 from the total exposure time this component, which was the integrated pulse times, was designated  $f_m$ . It is apparent that the ability of any frequency to produce injury is inversely related to the irradiation time required for damage so that an injury ability index may be designated to each frequency—merely the reciprocal of  $f_m$  multiplied by 10 to make the function a whole number.

Fig 2 gives the summary of the results and is a plot of injury ability against frequency. Injury ability is seen to increase rapidly below 1 MHz, reaches a relative plateau between 1 and 3.5 MHz and then rapidly decreases to approach zero before 5 MHz. The upper dotted line shows the thermocouple readings for the same irradiation regime and demonstrates that this is not a thermal effect since injury ability increases as thermal stresses decrease. These results were also confirmed by survival experiments when it was found that irradiation of the spinal cord at a frequency of 6 MHz for 15 min failed to produce paraplegia while irradiation for only 1 min at a frequency of 0.5 MHz was sufficient to produce a permanent paraplegia.

Next peak intensity was reduced from 50 to  $25 \text{ W cm}^{-2}$  thereby reducing time averaged intensity pro rata. This change had no effect on the injury ability of any frequency. It is likely that some threshold exists but this is difficult to determine owing to the complexity of the acoustic field. It is obviously of importance to establish the threshold for each frequency both for its clinical value and to indicate the mechanism of injury. As is shown later some diagnostic de-

Fig 2 The lower solid line is a graph of injury ability against frequency. The upper dotted line is a graph of temperature rise in spinal cord during irradiation at each frequency.

Fig 3 The lower solid line is a graph of injury ability against frequency when the animals breathed air. The upper dotted line is the injury ability against frequency when the arterial partial pressure of oxygen was 50 mm.

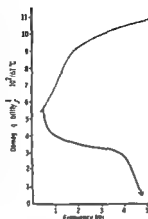


Fig 2

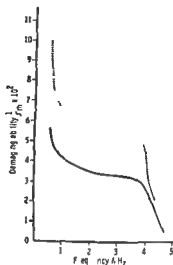


Fig 3

vices employ peak intensities considerably in excess of the  $25 \text{ W cm}^{-2}$  used in these experiments.

Several authors have reported that irradiation with ultrasound of frequency 1 MHz resulted in centrilobular necrosis (BELL 1957, CURTIS 1963, 1965). TAYLOR & POND (1970) confirmed this, and showed that this was not due to heat which produces perilobular necrosis. The latter authors therefore proposed that the relative hypoxia around the central vein may act as a synergistic injury factor with that of pulsed low frequency u/s. This was further investigated by rendering rats hypoxic and again determining the exposure time required to produce haemorrhage in the spinal cord. The results are summarised in Fig 3 which again is a plot of injury ability against frequency. The lower line shows the results obtained when the animals were breathing air while the upper one shows the results when the animals had an arterial oxygen partial pressure of only 50 mm. These graphs show that the presence of this degree of hypoxia increased the susceptibility of the tissue to injury by 40%. In fact there are a whole family of graphs whose separation from the parent line is proportional to the degree of hypoxia present.

One argument at present for the safety of diagnostic machines is that although they use high intensities in the pulses these are very short and thus cavitation is suppressed. Also the long intervals between pulses allow the induced heat to diffuse away. Heat and cavitation may thus be discounted. Such arguments do not apply to the mechanical effects of oscillatory forces where peak intensity may be paramount and time averaged intensity unimportant. Further experiments

Table

Frequency 3.5 MHz.  $I_p$  is peak intensity in  $W/cm^2$ . Pulsing is ratio of pulse length to intervals between pulses.  $I_a$  is time averaged intensity in  $W/cm^2$ .  $E$  is total exposure time in minutes.  $\int m$  is integrated pulse length during total exposure in seconds.

$I_p$	Pulsing	$I_a$	$E$	$\int m$
25	1/10	2.5	5	30
25	1/20	1.25	10	30
25	1/30	0.83	15	30
25	1/40	0.63	20	30

were therefore undertaken to establish the effect of decreasing the time averaged intensity by increasing the intervals between adjacent pulses. The results are summarised in the Table. A peak intensity of 25  $W/cm^2$  was used and delivery pulsed 1/10 giving a time averaged intensity of 2.5  $W/cm^2$ . A total exposure of 5 min was required to produce gross haemorrhage at this frequency of 3.5 MHz but since the delivery was pulsed 1/10, the actual dose received, the integrated pulse lengths ( $\int m$ ), was only 30 s. The intervals were then doubled to 1/20 reducing the time averaged intensity to 1.25  $W/cm^2$ . The total exposure time required then was 10 min but because of the increased interval, the accumulated dose time was still only 30 s. Further increasing the interval to 1/30 and 1/40 thereby reducing the average intensity to 0.63  $W/cm^2$ , linearly increased the total exposure time required but did not change the accumulated dose time of 30 s. Thus, despite the lowering of the time average intensity by increasing the interval between adjacent pulses the accumulated pulse lengths required for injury remained unchanged. Therefore, mechanical strains were accumulating at a constant rate and blood vessels ruptured when the same integrated dose had been received, which time was characteristic of each frequency and independent of the extent of the pulsing. More recent work has demonstrated the same accumulation of strains when two sub-threshold doses are separated by an interval of 30 min. Longer term accumulation has not yet been demonstrated.

The parameters used by diagnostic devices which are commercially available in Britain have been calibrated (HILL 1971). Peak intensity is of a special interest and may be seen to vary from 1.4 to 90  $W/cm^2$  in differing machines. Thus these machines are employing the same range of intensities and frequencies that were used in these experiments. When considering diagnostic machines a differentiation should be made between three modes of usage. First there is the use of repetitive pulses of very short duration but of high intensity. This may be called the sonar dose form for the demonstration of soft tissue structures. The second

mode is the use of low intensity continuous wave to recognise velocity by the change of reflected frequency which may be called the Doppler dose form. Finally there is the use of a high intensity pulsed beam, again for measuring velocity and this may be called the sonar Doppler dose form. The range of peak intensities used in these experiments is similar to those used in the sonar dose form but the pulsing regime lies between the sonar and Doppler dosage. When short pulses are used, and the shortest obtainable was 200  $\mu$ s injury still occurred but it took longer. Thus the evidence is that the shorter pulses are less injurious. Any relevance of these results to diagnostic usage therefore is only by extrapolation. Nevertheless, the demonstration of accumulation of mechanical strains and the synergistic effect of hypoxia are important findings. However, one may approximately calculate the safety limits of these machines on the basis of these experiments, this is, of course, only for this injury factor.

If long term accumulation occurs as it did in these shorter term experiments, and assuming that the diagnostic pulses are no less injurious than the shortest pulse reported here of 200  $\mu$ s, then even if the tissue were highly hypoxic it would still require a continuous exposure of approximately 3 to 6 h before injury could be expected. Since in the normal scanning procedure, any organ receives only a transient exposure it would appear that the ultrasound, as currently used, has an enormous margin of safety. However, other forces are being exerted and each of these must be defined and their biologic effects determined. In particular toxicity studies on the Doppler dose form must be carried out so that long term foetal monitoring may be performed with confidence.

### Acknowledgements

We would like to thank Professor Warwick for his advice and encouragement, Mr Raymond Wright for technical assistance and Mr N. Fitzpatrick for photography.

### SUMMARY

Haemorrhage into the spinal cord of the rat was used to compare the effects of ultrasonic irradiation of varying parameters. The ability to produce injury was found to decrease with increasing frequency. The threshold intensity for this mechanical injury factor is less than 25 W cm<sup>-2</sup> peak power. Short term accumulation of mechanical strains from ultrasonic stress was demonstrated. Hypoxia was shown to be a synergistic factor with the mechanical injury agent. When these results are extrapolated to diagnostic pulsing regime a large safety margin is indicated for the present conventional use of diagnostic ultrasound.

### ZUSAMMENFASSUNG

Eine Blutung in das Rückenmark der Ratte wurde verwendet um die Wirkung von Ultraschall Bestrahlung verschiedener Eigenschaften zu vergleichen. Es wurde gefunden, dass der schädigende Effekt mit steigender Frequenz abfiel. Die Grenzwertsintensität für

diesen mechanischen Schädigungsfaktor ist niedriger als  $25 \text{ W cm}^{-2}$  Spitzenleistung. Kurzzeitige Akkumulation von mechanischen Spannungen durch Ultraschallstress wurde demonstriert. Es wurde gezeigt, dass Hypoxie synergistisch mit dem mechanischen schädlichen Agens wirkt. Wenn diese Ergebnisse auf das diagnostische Pulsationssystem extrapoliert werden, ergibt sich ein weiter Sicherheitsabstand für den gegenwärtigen konventionellen Gebrauch diagnostischen Ultraschalls.

## RÉSUMÉ

Les auteurs ont utilisé l'hémorragie dans la moelle épinière du rat pour comparer les effets d'irradiations par les ultrasons en fonction de la variation des paramètres. Ils ont constaté que l'élévation de la fréquence diminue la nocivité de l'irradiation. L'intensité seule pour ce facteur de lésion mécanique est une puissance maximale inférieure à  $25 \text{ W cm}^{-2}$ . Ils ont mis en évidence la summation à court terme de contraintes mécaniques provoquées par l'agression ultrasonique. Ils ont montré que l'hypoxie est un facteur synergique de l'agent lésionnel mécanique. Si on extrapole ces résultats au régime pulsatoire de diagnostic, on constate que l'utilisation habituelle des ultrasons pour le diagnostic comporte une grande marge de sécurité.

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**SPINE**





## DIMER X

### A new contrast medium for lumbar myelography without spinal anaesthesia

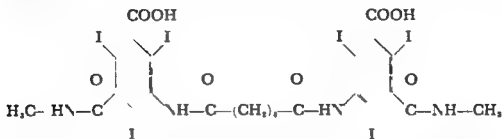
by

P. AHLGREN

Practically all lumbar myelographies have been performed for many years at least in the Scandinavian countries with the water soluble contrast medium Na monoiodomethane sulphonate (Methiodal, Conturex, Kontrast U, Abrodil). ARNEIL & LIDSTROM published accounts of their first experiments in 1931, but not until after 1944 has this method been more widely employed. Since then numerous papers have been published on complications either caused directly by the contrast medium or indirectly by the spinal anaesthesia which was an absolute prerequisite (AHLGREN & PRÆSTHOLM 1969).

A report by CAMPBELL *et al.* (1964) on the employment of methylglucamine iothalamate in lumbar myelography without spinal anaesthesia (Conray Meglumun 282, Conray 60, Contrix 28) but in particular after more recent publications from France and Belgium (5, 6, 7, 13, 14, 20, 21) and Scandinavia (1, 12, 19, 22) Methiodal appears to have been replaced by the iothalamate. All the reports disclose the fact that complications may occur during and after myelography with iothalamate, and although these are usually of a mild nature only deaths have also been reported (19). Conray Meglumun thus appears

to be a more satisfactory contrast medium than Methiodal although a still better preparation would be acceptable. GONSETTE & ANDRE BALISAUX (1969) reported at the XII International Congress of Radiology Tokyo 1969 and more recently (1970) on the toxicologic and clinical effects of methylglucamine iocarmate, which is at dimerisation of iothalamate. Dimer X is an aqueous solution of the meglumine salt. Salt content 60%, iodine content 28%. It has the formula



GONSETTE & ANDRE BALISAUX stated that this contrast medium was considerably less toxic and in particular less neurotoxic than the basic product Conray. Meglumine HAYES et coll (1966), HALAL (1966) and BJORK et coll (1969) also reported that the dimerised products of the contrast media were less toxic for angiography.

*Material* A total of 200 myelographies with Dimer X were carried out during the period November 1969–August 1970 and compared with 408 Conray myelographies performed during the period June 1968–November 1969. Five Conray and three Dimer X myelograms failed in the first instance because of the total or subtotal subdural accumulation of the contrast medium, but were successfully repeated later. The sex and age distribution was almost identical in the two series.

*Technique* Identical techniques were employed with both contrast media for the purpose of comparison, the dose was 7.5 ml diluted with 2.5 ml of spinal fluid. Physiologic saline was used for dilution in a few patients but this always resulted with both contrast media in less distinct outlines. The spinal puncture and the injection of contrast medium were made with the patient lying on the left side with the head of the table elevated 15°. A suitable dose of contrast medium (cf Table 4) was injected during TV fluoroscopy with the object of obtaining a diagnostic level of the contrast column up to and including the third lumbar disc. The rate of injection should not be too rapid as the contrast medium will then be boosted to excessive levels with the danger of spasm. The latter has been reported from another hospital in the first 6 patients

Table 1

Reactions (in per cent) during the examinations. None of these reactions necessitated treatment except injection of ephedrine in two patients with fall in blood pressure

Reactions	Dimer X	Conray
Slight tension in back or legs	10.5	51.5
Accentuation of usual pain	7.5	19.0
Other kinds of pain	0	3.4
Tonic spasm	0	0.5
Fall in blood pressure $> 10$ mm	9.0	12.7
(10 mm $< BP < 30$ mm)	(7.0)	(9.5)
(30 mm $< BP$ )	(2.0)	(3.2)
Rise in blood pressure $< 10$ mm	4.0	1.2

undergoing Conray myelography and resulting from too rapid injection of the contrast medium. Changes in the technique and injection rate have led to symptom free examinations (PRESTON & LESTER 1970).

Films were taken prone in the lateral and oblique positions with 20° and 30° elevation first of the diseased and then of the healthy side with a vertical beam direction and then also in the right and left lateral positions with a horizontal beam direction. If the roots were poorly filled films were taken with the patient erect in an attempt to obtain better demonstration of possible normal roots if required the examination was supplemented by examination of function in an attempt to reveal clinically distinct but myelographically doubtful prolapses.

Premedication is not essential but experience has shown that if 25 mg of promethazine are injected intramuscularly thirty minutes before the examination the patient will be sufficiently tranquil without being drowsy, and the blood pressure will not be affected. Diazepam is useless as a prophylaxis against possible pains which if they occur at all will not do so until its effect has subsided. Diazepam in a few hospitals has produced a higher incidence of fall in blood pressure than that observed later when its use was abandoned.

The only difference in the procedure when employing the two substances is the position of the patient after the examination. Following Dimer X myelography the body should be kept raised between 10° and 15° for six hours. After Conray myelographies the body must be kept elevated about 70° for six hours and the patient must be absolutely calm and relaxed. It has been observed that the incidence of spasm was considerably higher in those patients who for the purpose of checking the absorption rate were brought back to the roentgen department between one and four hours after the injection of the contrast medium.

Table 2

*Reactions (in per cent) during the first 72 hours after the examination*

Reactions	Contrast medium	Day of examination	Second day	Third day
Headache	Dimer X	18.0	24.0	14.0
	Conray	26.0	43.0	11.2
Dizziness	Dimer X	5.0	5.5	3.0
	Conray	4.5	11.0	1.5
Nausea	Dimer X	3.0	2.5	2.5
	Conray	5.9	1.9	1.5
Vomiting	Dimer X	0.5	0.5	1.0
	Conray	4.1	0	0
Usual pain	Dimer X	80.0	51.0	53.0
	Conray	73.6	71.4	62.8
Accentuated pain	Dimer X	13.0	12.0	8.0
	Conray	11.5	7.4	4.8
Other pain	Dimer X	4.5	10.0	7.3
	Conray	10.0	8.4	7.1
Raised temperature	Dimer X	8.0	15.0	3.0
	Conray	7.6	15.4	3.7

*Reactions during the examination* No particularly troublesome complications occurred during the examination itself (Table 1). Some patients complained of slight tension in the back during the actual injection, perhaps radiating to the leg on which the patient was lying and occasionally accompanied by mild sensations of formication. Pain already present might be aggravated but never to such a degree that analgetics were required. Actual pain in the back or pain radiating to the healthy leg or to other regions has occurred. Increased muscular tone or muscular fibrillations were recorded in two patients in whom Conray was employed, however only as symptoms and not as signs. A fall in blood pressure  $\geq 10$  mm was recorded in a few instances but only occasionally did this not become normal spontaneously, so that ephedrine was required. The rise in blood pressure was undoubtedly caused sometimes by anxiety.

*Reactions after the examination* The patients were interviewed daily for three days after the examination and the most important of numerous routine questions are listed in Table 2. The greatest difference between Dimer X and Conray appears in the number of patients complaining of headache. The considerably lower frequency after Dimer X was caused no doubt by the less elevated position after the examination resulting in a smaller amount of diffusing spinal fluid. The higher frequency on the second day was due to the fact that all the patients from the neurosurgical ward are allowed out of bed

Table 3

*Spasms correlated to the diagnostic contrast level*

	No of examinations	Th12	L1	L2	L3	L4
Dimer X	197	4	45	86	48	4
Clonic spasm	9		3	4	2	
Percentage	4.6		6.7	4.6	4.2	
Conray	403	3	37	130	209	20
Clonic spasm	18	2	4	7	4	
Percentage	4.4	(40)	10.8	5.4	1.9	

on the morning after the examination and a few of them on the day of examination while the patients in the departments of physiotherapy and neurology are kept in bed until the third day or later.

As regards late effects and slight symptoms no greater difference in frequency was observed between Dimer X and Conray although on the whole these side effects were less frequent after the former. Aggravation of existing pain is an exception in that Dimer X presents a slightly higher frequency. It is not easy to explain this phenomenon because the number of patients complaining of pain already present was considerably lower on the second and third day. The temperature in some of these measured in the morning and evening was only 0.5° C above normal levels and usually only between 0.1 and 0.2° C higher at one or two measurements. Stiffness of the neck was never recorded.

*Clonic spasm.* The contrast media produced one common complication. In both series clonic spasms were observed in about 4.5 per cent of the patients commencing from 2 to 9 hours after injection of the contrast medium generally about 4 hours after Conray and 6 hours or later after Dimer X. The intensity and duration of the spasms appear to be independent of the type of contrast medium and in the 27 patients were alleviated after one intravenous injection of 5 to 10 mg Valium in 11 patients and after two injections in 9 patients. 3 patients did not find it necessary to ask for drugs because the spasms were only slight. The spasms in 2 patients were not relieved by Valium and did not cease until pethidine or tetrapone had been given. Two patients had persistent spasm resistant to treatment for about 9 hours; the spasms in one patient were only slight and occurred after Dimer X and in another patient they arose after Conray and were tonic and clonic and of such severity that decubitus ulcer developed on the medial side of the knees and the malleoli.

One patient with spasm after Conray was free from any symptom or sign after subsequent Dimer X myelography in spite of the fact that the Dimer X

Table 4

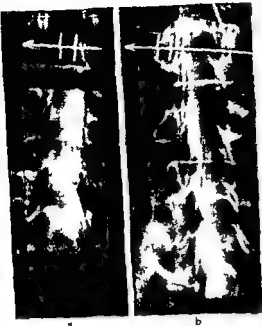
*Spasms correlated to the injected volume of a mixture of contrast medium and spinal fluid. One patient not included in the table received 13 ml Dimer X because of an extraordinarily large sacral sac; spasms occurred eight hours after injection. Up to 16 ml Conray has been used in similar instances but part of it has been aspirated after the examination; no spasms occurred after these myelographies.*

	< 7 ml	7 ml	8 ml	9 ml	10 ml
Dimer X	10	18	30	118	90
Clonic spasm	1		1	6	
Percentage	(10)		3.3	5.1	
Conray	24	20	79	129	153
Clonic spasm			2	6	10
Percentage			2.5	4.7	6.5

contrast column was one segment higher than with Conray. One of the patients with spasm had undergone Conray myelography at another hospital and two Dimer X myelographies at our hospital on all three occasions resulting in clonic spasm. However, at no time did the patient ask for Diazepam although she had been informed in advance that the drug was available. She stated that she had had déjà vu and petit mal type seizures but had never had grand mal attacks or received antiepileptic treatment. EEG revealed no epileptic changes, but only those produced by drugs.

Two patients with verified epilepsy, well controlled on antiepileptic drugs, failed to develop spasm following myelography with Conray or Dimer X. Hence, in patients with epilepsy the absolute contraindication to myelography with water soluble contrast media may be of a relative nature only although, until further proof is available, the indication for myelography in epileptic patients must be rather stronger than with other groups of patients.

The spasms are correlated to the diagnostic level of the contrast column in Table 3 and the amount of contrast medium injected in Table 4. A distinct correlation is evident between increased frequency of spasm and both high diagnostic and maximum levels of the contrast column and to a large dose of the medium. The maximum level of contrast medium is practically always one segment higher than the diagnostic level. This applies to the entire Conray series whereas the maximum and the diagnostic levels of the contrast medium are almost identical in the latter two thirds of the Dimer X series. The level is increased in this last group in spite of the application of the same dose of contrast medium because of the possibility of exposures with a horizontal beam direction with the patient lateral (see Figure). However, this does not occur regularly, because a slow rate of injection produces sedimentation of the heavy medium.



Myelography with 9 ml Dimer X a) Prone position vertical beam direction. b) Lateral decubitus horizontal beam direction. The same diagnostic and maximum contrast levels in (b) but one segment lower diagnostic level than maximum contrast level in (a)

and equally low diagnostic and maximum levels. Rapid injection results however in a greater difference between the diagnostic and maximum levels as well as in poor contrast since the medium diffuses too much with the spinal fluid during the injection.

### Conclusion

The introduction of Conray myelography has meant an enormous advance in the diagnosis of lumbar disc herniation. An evaluation of Dimer X suggests further progress although even less toxic water soluble contrast media which could be employed throughout the spinal column would be desirable. The few and slight clonic spasms which lend themselves easily to treatment demand individual dosage aiming at a diagnostic level to include that of the third lumbar disc. A mixture of the contrast medium and spinal fluid in the ratio 3:1 produces the best contrast and the highest diagnostic accuracy. Because clonic spasm still occurs in about 45 per cent of examinations Dimer X does not permit its application higher than the first or second lumbar discs any more than Conray does.

The identical frequencies of spasm after myelography with Dimer X and Conray, in spite of the lower neurotoxicity demonstrated experimentally, are



presumably caused both by the change in position following the myelography and by the absence of routine on the part of examiners during fluoroscopy at the level of the contrast column. It has been possible from the films to predict the occurrence of spasm in many of the patients in which this has actually occurred. Inexperienced examiners must be carefully instructed and there must also be a standing rule that the examination technique is constantly supervised and corrected. In examinations following which emergency surgery can be expected the lumbar needle must remain in position throughout and as large a portion as possible of the contrast medium must be aspirated at their conclusion. Two of the aforementioned patients who developed spasms, one after Conray and the other after Dimer  $\lambda$ , underwent emergency surgery and the spasms occurred when they became conscious after the operation. In neither of these was the contrast medium aspirated. Routine aspiration and sometimes, washing out with saline, as reported from another hospital appear to be an unnecessary procedure and, furthermore, give rise to an undue risk of infection.

The intensity of the two contrast media is identical although Dimer  $\lambda$  seems to have a slightly higher viscosity and consequently, when used, more films have to be taken with the patient standing than necessary with Conray.

## SUMMARY

A total of 408 lumbar myelographies with Conray Meglumine 282 and 200 with Dimer  $\lambda$  using the identical technique are compared. The reactions are rather less after Dimer  $\lambda$  than after the former although the tendency to produce spasms is the same with the two contrast media. It is suggested that Dimer  $\lambda$  should replace Conray.

## ZUSAMMENFASSUNG

Insgesamt 408 lumbale Myelographien mit Conray Meglumine 282 und 200 mit Dimer  $\lambda$  unter Verwendung identischer Verfahren werden verglichen. Die Reaktionen nach Dimer  $\lambda$  sind eher geringer als nach Conray obwohl die Tendenz, Spasmen hervorzurufen die gleiche für beide Kontrastmittel ist. Es wird vorgeschlagen dass Dimer  $\lambda$  Conray ersetzen sollte.

## RÉSUMÉ

L'auteur compare un total de 408 radiculographies lombaires faites avec Conray Meglumine 282 et 200 radiculographies faites avec Dimer  $\lambda$  suivant la même technique. Les réactions sont plutôt moindres après Dimer  $\lambda$  qu'après Conray Meglumine cependant la tendance à produire des spasmes est la même avec ces deux moyens de contraste. L'auteur pense que le Dimer  $\lambda$  devrait remplacer le Conray.

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## UNUSAL MYELOGRAPHIC APPEARANCES OF THE LUMBOSACRAL RADICULAR SHEATHS

by

M. CASTRO and J. TORRES

Lumbosacral pain syndromes may arise from causes other than disc herniation, among which congenital cysts of the spinal membranes play an important role. Their clinical signs and radiologic diagnosis should present no real difficulties. A number of unusual appearances in the lumbosacral region during myelography for disc lesions or congenital cysts of the lumbosacral root sheaths led to an investigation to establish their possible nature. The literature seems to contain no record of similar work; the possibility of the appearances being due to artefacts caused by technical errors has been eliminated.

The records of 9 patients who had had a lumbosacral pain syndrome or mild motor impairment of the lower limbs were analysed. All the patients underwent myelographic examination with the positive contrast medium Lipiodol ultrafluide injected into the spinal canal through an atraumatic lumbar puncture, the average amount injected being 10 ml. The examination was performed with TV fluoroscopy on a tilting table with the patient prone, at its conclusion most of the contrast medium was removed by means of a further tap. None of the patients had had previous lumbar puncture; spinal fluid was always withdrawn at the time of the examination for analytic purpose and before the injection of the contrast medium.



Fig 1 Oil myelography in a female aged 24 with lumbosacral pain and bilateral motor impairment of the lower extremities for two months. Weight loss ESR + a) Patient erect immediately after injection b) At 20 minutes Filling of lumbar and sacral root sheaths beyond the spinal ganglion Pseudocystic dilatations in lower lumbar radicular pouches c) At 24 hours The contrast medium has reached the perineurial space

The age sex and symptomatology of the patients are given in the Table. The dominant symptom was lumbago in those patients that had had pain; the other group with motor impairment had had no pain. Two of the 6 patients with pain had disc herniations demonstrated radiologically; the symptoms ended when the discs were removed. The other patients were treated conservatively (e.g. anti-inflammatory therapy, bed rest). All improved.

The appearances at myelography were constant (Fig 1) with rapid and complete filling of the irregular nerve root sheaths up to the spinal ganglion and fairly rapid outlining of the peripheral nerve after some minutes (not more

Table  
Age, sex and symptomatology of patients

Age (years)	Sex	Duration of symptoms or signs	Lumbosacral pain	Motor impairment
30	♀	3 years	—	—
41	♀	20 days	+	+
37	♂	3 days	+	—
61	♀	4 years	—	+
24	♀	3 years	+	+
29	♂	19 months	+	—
18	♂	3 years	—	+
27	♂	2 months	—	+
16	♂	2 years	+	—



Fig. 1 Oil myelography in a female aged 28. Severe thoracic-abdominal trauma three years previously (FSR +). a) Immediately after injection. Widened radicular pouches corresponding to L3 and L4 bilaterally and left L5 root. b) At 15 minutes. Contrast medium lies along the perineural space beyond the paral ganglia of the L4 roots. c) At 24 hours. The contrast medium has now reached the perineural spaces of the L4, L5 and S1 roots.

than fifteen minutes). The contrast medium flowed freely into the subarachnoid space along the nerve beyond the paral ganglion when the table was tilted (Fig. 2). Roentgenograms obtained 24 or more hours later revealed further passage of the medium in 4 patients (Fig. 3). The width of the radicular sheaths was usually normal or slightly increased but pseudocystic dilatations resembling perineural cysts were evident in the lumbosacral region in 3 patients.

The spinal canal of children still born at term was injected with thorium dioxide (Thorotrast). The amount (3 to 5 ml) varied according to the weight of the child and ap films were obtained. Appearances varying between the normal and those suggesting the presence of meningeal diverticula were evident in these bodies. Similarities with the myelographic appearances described in the present paper were not evident.

### Discussion

The radiologic literature appears to contain no description of this abnormality. SOLT LIFMAN (1963) writing on perineural sacral cysts described appearances similar to those now presented. He referred only to marked filling of the radicular sheaths without investigating its possible nature and etiology, in an asymptomatic patient. ITHUR reported the same appearances in the lumbosacral region in patients in whom an oily contrast medium had been introduced into the lateral ventricles for stereotaxic purposes. NAGEOTTE (1902) published his observations on the pathologic aspects of endoneuritis and perineuritis of the



Fig 4 Cavitation in central part of a fasciculus of a posterior root in radicular neuritis of undetermined origin (Reproduced from C R S & Biol 54 (1907) 1443)



Fig 3 Myelography in a male aged 39 with left lumbosacral pain for a year. a) Immediately after injection. Slight filling of the left L4 radicular sheath. Compression of the left L5 radicular pouch (disc herniation). b) At 20 minutes and after removal of some of the contrast medium. Filling of the radicular sheaths and perineurial spaces of the left L4 and L5 roots with cystic dilatation of the latter. c) At 72 hours. The contrast medium now lies along the perineurial spaces of the left L5 and sacral roots.

nerve roots as well as the spinal ganglion with the formation of cystic cavities (Fig 4). REYED (1947, added further information and later (1959) analyzed 26 cases in 10 of which NAGEOTTE's findings were confirmed. The appearances are different from those in perineurial cysts and approach more to those of normal radicular pouches. The nerve sheath in the present material was pathologically enlarged and irregular in appearance. The changes would seem

to indicate nerve root compression. As with perineural cysts it may be that inflammatory processes or small haemorrhages provoked by small repeated traumas may be responsible for the arachnoidal proliferation with cystic formation.

### Acknowledgement

The authors take this opportunity of thanking the Obstetric Service of the Hospital del Salvador (Director Prof. L. Tisne) for affording them access to the still born children.

### SUMMARY

Unusual appearances observed in lumbar myelographies for disc lesions or congenital cysts of the root sheaths have been investigated. Their nature is considered and discussed; they are not due to artefacts.

### ZUSAMMENFASSUNG

Ungewöhnliche Erscheinungen, die bei lumbalen Myelographien von Diskus-Schaden oder kongenitalen Cysten der Wurzelscheiden beobachtet wurden, wurden untersucht. Deren Natur wird erörtert und diskutiert; sie sind nicht Folge von Artefakten.

### RÉSUMÉ

Les auteurs ont étudié les aspects inhabituels observés au cours de myélographies lombaires pour des lésions de disques ou des kystes congénitaux des gaines radiculaires. Ils étudient leur nature. Ils ne sont pas dus à des artefacts.

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## DEVELOPMENT OF SPINAL CORD ANGIOGRAPHY

by

GIOVANNI DI CHIRO

The technique of spinal cord angiography is rapidly becoming one of the essential procedures in the neuroradiologic armamentarium. It is therefore important at this time to outline the various phases of development of this diagnostic method.

Occasional reports of angiographic demonstration of arteriovenous malformations of the spinal cord may be found already in the literature of the 50's. The first is probably the case of HENSON & CROFT who reported in 1956 an arteriovenous malformation of the cervical cord which had been demonstrated, back in 1953, by vertebral angiography. RAND & RAND (1960) in the chapter of their textbook *Intraspinal tumors of childhood* dedicated to vascular anomalies of the spinal cord described an arteriovenous malformation which had been demonstrated by abdominal aortography in 1954. Additional cases of arteriovenous malformations of the cervical cord investigated by vertebral angiography were reported in 1958 by HOOK & LIDVALL and in 1960 by MORRIS. The first angiographic demonstration of a spinal cord tumor was reported by DI CHIRO in 1957.

In 1962 DJINDJIAN and co-workers reported their first angiographic investigation of an intraspinal arteriovenous malformation and initiated a systematic



First selective angiography of spinal cord carried out in December 1964. Lower arrow points to tip of catheter in left 17th intercostal artery and upper arrows to part of malformation. (Residual) Pantopaque from previous myelography.



angiographic exploration of the arteriovenous malformations of the spinal cord. This led to the publication in 1966 of a monograph by DJINDJIAN *et coll.* which included a thorough analysis of 12 cases of intraspinal arteriovenous aneurysms. These French authors emphasized the importance of subtraction for their angiographic investigations.

In 1964 our group in Bethesda started a program dealing with angiography of the arteriovenous malformations of the spinal cord. In the beginning we concentrated our attention on subtraction angiography (DOPPMAN & DI CHIRO 1966). We soon, however, realized that new approaches were indispensable. The improvement offered by subtraction was not enough to insure the demonstration of the spinal cord vessels during midstream aortography. The next logical step was to perform selective angiography of the spinal cord.

In December of 1964 we carried out the first successful selective angiography of the spinal cord by injecting contrast medium in the intercostal arteries

(Figure). During 1965 we expanded our experience and we reported on this at the Thirty-fourth Annual Meeting of the Harvey Cushing Society in St. Louis, April 1966 and at the Fifty-second Annual Meeting of the Radiological Society of North America in Chicago in November of the same year. In 1967 we published a report on our investigation of 12 cases which included control

selective angiography after ligation of the malformation feeders in four patients (Di Cenzo et coll.) In this article we also commented on our experience with selective angiography of the spinal cord in normal cases and in patients with pathology other than arteriovenous malformations such as tumors, syringomyelia and secondary dilatation of the spinal cord vessels.

After this 1967 report, a large number of publications have appeared in the American and European literature dealing with spinal cord angiography. The selective injection of the intercostal and lumbar arteries has made the difference between an occasional demonstration and a reliable consistent demonstration of the spinal cord vessels in the thoracolumbar cord. A frequent but not constant, demonstration of the cervical and high thoracic cord vessels is obtained by selective injection in the vertebral arteries, costocervical trunks, and high intercostal arteries.

Selective angiography of the spinal cord has been shown to be a safe technique possibly safer than midstream aortography. We are unaware of any significant complication caused by this procedure. The probable explanation for the high safety of the selective technique is that by this method we do not flood simultaneously all the vessels from which the radicular branches reaching the cord originate. A rapid washout of the contrast filled spinal cord arteries by the blood rushing from neighbouring vessels which do not contain contrast medium is thus possible.

Selective angiography of the spinal cord is a somewhat complex and time-requiring technique. We are therefore exploring new approaches in experimental models (Di Cenzo et coll. 1970) and in humans. Two modifications of spinal cord angiography—pressor amine potentiation and abdominal compression—have proven very successful in the monkey. It remains to be seen whether or not the new approaches will be transferable to human clinical material. In the meantime selective angiography must remain the procedure of choice.

## SUMMARY

A short outline of the development of spinal cord angiography with particular emphasis on the importance of the selective technique.

## ZUSAMMENFASSUNG

Eine kurze Darstellung der Entwicklung der Angiographie des Rückenmarks unter besonderer Berücksichtigung der Bedeutung der selektiven Technik.

## RÉSUMÉ

L'auteur fait un bref historique du développement de l'angiographie de la moelle épinière et insiste particulièrement sur l'importance de la technique sélective

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## ANGIOGRAPHIE DE LA MOËLLE EPINIÈRE

Acquisitions et orientations de recherche au  
terme d'une expérience de 10 ans

par

R. DJINDJIAN, M. HURTH et R. HOUDART

Depuis 1961 nous avons réuni (au 1<sup>er</sup> 1970) 75 observations de malformations vasculaires médullaires. Tous ces cas concernent des malformations artériovoineuses à l'exclusion de toute autre variété. Pendant cette même période près de 400 explorations angiographiques à visée médullaire ont été réalisées et jamais dans le domaine des malformations vasculaires nous n'avons rencontré d'autre lésion que l'angiome. Ceci permet donc de penser qu'au niveau de la moëlle épinière la malformation artériovoineuse représente le type de malformation sinon exclusif du moins de beaucoup le plus fréquent. L'anévrisme artériel pur (dont la réalité peut être mise en doute) les télangiectasies apparaissent ainsi comme infiniment rares et dans l'état actuel des techniques échappent à l'angiographie.

Les malformations artériovoineuses médullaires présentent un polymorphisme morphologique indiscutable. Leur mode d'alimentation artérielle (artères tributaires du système spinal antérieur ou du système spinal postérieur) conditionne leur situation intra médullaire, extra médullaire ou mixte et du même coup leur opérabilité. Leur volume semble en grande partie lié à la pluralité des afférences artérielles et il est évident qu'ici pour des raisons embryologiques (modalités chronologiques d'évolution du réseau vasculaire médullaire qui progresse dans

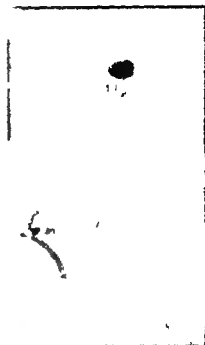


Fig 1 Malformation à composante artérielle. Artère nutritrice naissant de la quatrième artère intercostale droite injectant un lac vasculaire d'aspect anévrysmal.

le sens cranio caudal et d'avant en arrière), la topographie en hauteur de la malformation conditionne en partie sa morphologie.

Cependant si la présence de shunt artério-veineux constitue le commun dénominateur de ces malformations leur morphologie est à côté des facteurs liés aux modalités de l'alimentation artérielle également dépendante de l'importance du drainage veineux.

Tous les intermédiaires sont possibles entre les cas où la composante artérielle (Fig 1) est prédominante et ceux où la composante veineuse est au premier plan (Fig 2).

Il est ainsi très surprenant de découvrir certaines malformations de la jonction dorso-lombaire dont le drainage veineux peut être suivi jusqu'au niveau des systèmes veineux de la fosse postérieure voire des veines cérébrales profondes.

De tels faits ne doivent rien changer à notre avis à la conception générale des lésions anormales congénitales comportant un défaut de maturation du système vasculaire avec défaut d'élaboration du lit capillaire et persistance de fistules artério-veineuses mais expliquent probablement certaines descriptions antérieures (angiomes artériels et angiomes veineux). Plutôt qu'une évolution secondaire de la malformation après la naissance avec dilatation évolutive des voies de drainage nous serions enclin à penser que ces formes à composante veineuse



Fig 2 Malformation a composante veineuse (a terminaison dans la veine cave inferieure) a) Malformation dorso-lombaire nourrie par la première artère lombaire gauche b) Il se termine dans la veine cave inferieure par l'intermédiaire des artères sacrées latérales

dominante reflètent un balancement possible dans la malfaçon qui affecte ici au premier plan le système artériel et là le système veineux mais comporte toujours une communication pathologique entre ces deux systèmes

### Progres recents du bilan angiographique

*L'angiographie sélective de la moelle cervicale* (Fig 3) La nécessité et l'intérêt du cathétérisme sélectif des afférences artérielles médullaires pour l'étude angiographique des malformations artério-veineuses spinales constituent deux notions solidement établies

Réalisée dès 1966 à l'étage des artères intercostales et lombaires l'angiographie sélective des branches du système sous-clavier destinées à la moelle cervicale n'est pas devenue pour nous de pratique courante que depuis 1968



Fig. 3. Angiographie cervicale sélective (chez un enfant). a) Angiographie sous clavière gauche par voie fémorale. b) Angiographie sélective de l'artère vertébrale gauche opécifier le pédicule de l'arc postérieur de l'anneau arrière du renflement cervical)

Les avantages de la technique d'injection sélective par les procédés globaux sont certains. Précision du diagnostic — la finesse de la méthode fait pratiquement totalement disparaître le risque de méconnaître une malformation de petit volume. Précision du bilan morphologique — l'étude successive et sélective des différents pédicules de la malformation donne un reflet très fidèle de leur nombre et ce qui est essentiel pour le traitement de leur situation antérieure ou postérieure. De même les rapports avec le réseau fonctionnel de la moelle elle-même peuvent être établis puisque l'angiographie sélective en permet l'opacification.

*L'angiographie sélective chez le jeune enfant.* Ce procédé est dans notre expérience utilisable à partir de l'âge de 3 ans (le problème ne s'est jamais posé pour nous au dessous de cet âge). Il implique l'utilisation de sondes fines, une particulière douceur dans les manipulations et bien sûr la réduction des quantités de produit de contraste injectées. Comme chez l'adulte et le grand enfant, le cathétérisme sélectif des artères à destination médullaire accroît considérablement la fidélité, la précision et la signification thérapeutique du bilan angiographique des malformations à résection clinique précoce. Cette éventualité n'est pas exceptionnelle puisque notre série de 75 malformations médullaires comporte 23



Fig 4 Angiographie des arteres centrales et sulco-commissurales (→) dilatees nourrissant une malformation dorso-lombaire



Fig 5 Malformation dorsale retro-medullaire avec un pedicule nourricier stenose (→) (cinquieme artere intercostale gauche)

observations de sujets ages de moins de 15 ans lors de l'installation des premiers symptomes de la maladie et 5 d'entre eux n'avaient pas depasse 6 ans

*Mise en evidence angiographique des arteres centrales et sulco commissurales* (Fig 4) Ces arteres de tres fin calibre (16 a 32  $\mu$ ) naissent de l'axe spinal anterieur et se distribuent schematiquement aux trois-quarts anterieurs de la moelle. En l'absence de dilatation pathologique elles demeurent angiographiquement invisibles. Leur augmentation de calibre s'observe lorsqu'elles participent a la vascularisation de certaines tumeurs (angioréticulomes) ou encore des malformations a composante intra medullaire. Il est alors possible de les identifier lors de l'angiographie. Cette mise en evidence necessite la prise de clichés tres precoces, la multiplication des incidences (profils et obliques) et l'aide d'agrandissements. Le probleme n'est pas uniquement theorique si l'on doit un jour envisager l'abord chirurgical de telles arteres (voies donnant acces a la face anterieure de la moelle, microscope operatoire et coagulation bipolaire) pour le traitement des malformations intra medullaires.



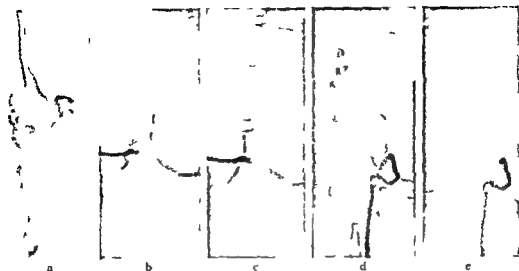


Fig 6 Malformation mixte dorso-lombaire multi pédiculaire (embolisation partielle de l'afférence postérieure) a) Portion intra médullaire de la malformation irriguée par l'artère d'Adam Kiewicz issue de la neuvième artère intercostale gauche b) c) Portion retro-médullaire irriguée par la neuvième artère intercostale droite avant (b) et après (c) embolisation d) e) Portion retro-médullaire irriguée par la première artère lombaire gauche avant (d) et après (e) embolisation

### Les limites du traitement chirurgical

L'angiographie conditionne totalement le traitement des malformations médullaires en définissant l'appartenance des pédicules artériels aux axes spinal antérieur ou spinaux postérieurs et donc le siège juxta ou intra médullaire de la malformation.

Les malformations exclusivement alimentées par des artères du système spinal postérieur sont toujours dans notre expérience extra médullaires, postérieures et totalement extirpables (Fig 5). L'exérèse chirurgicale représente alors la solution thérapeutique de choix et demande toujours à être contrôlée par une angiographie post-opératoire. Celle-ci permettra dans les cas privilégiés de juger de la réapparition après ablation de la malformation du réseau fonctionnel médullaire le plus souvent invisible avant l'intervention. L'angiographie de contrôle apporte dans ces cas un argument indirect à la théorie du vol vasculaire qui fait jouer à l'ischémie par hémodétournement le rôle principal dans la genèse de l'atteinte médullaire des malformations. C'est la suppression chirurgicale des shunts artério-veineux qui permet l'injection post-opératoire du réseau artériel normal.

Les malformations mixtes (Fig 6) alimentées par les deux réseaux antérieur et postérieur comportent toujours une portion intra médullaire chirurgicalement intouchable c'est à dire que leur traitement est toujours incomplet. Lorsque la portion postérieure de la malformation est prédominante il semble légitime de

tenter l'exérèse subtotale. C'est opératoirement que l'on jugera des limites à assigner à l'acte chirurgical. Dans un certain nombre de cas les portions intra et extramedullaires de la malformation paraissent indépendantes ce qui permet sans difficulté technique insurmontable l'ablation des neo-vaisseaux postérieurs. Quand l'alimentation antérieure représente la source essentielle de vascularisation de la malformation l'embolisation musculaire des afférences postérieures constitue une thérapeutique à la fois élégante, atraumatique et au moins temporairement efficace.

Le problème majeur demeure celui de l'attitude à adopter vis-à-vis des afférences antérieures. Lorsqu'angiographiquement la malformation apparaît directement alimentée par une artère médullaire majeure (artère du renflement cervical ou artère du renflement lombaire), il ne nous semble pas licite de proposer son interruption. La question est comment on peut suivre les auteurs qui considèrent une telle artère comme entièrement malformative alors que visiblement elle représente la source essentielle de vascularisation médullaire dans la zone de la malformation. Nous ne reconnaissons ni les possibilités de suppléance parfaitement étudiées anatomiquement par LAZORTHES & GOUAZE (1968) et récemment concrétisées chez le singe par les travaux de FRIED et coll (1969) ni les rares observations d'interruption bien tolérée de pédicules antérieurs (NEWTON & ADAMS 1968, DOPPMAN et coll 1968). Il nous paraît dangereux cependant de proposer sans préalable l'interruption des afférences antérieures des malformations. Des recherches complémentaires sont nécessaires dans ce domaine et pour notre part nous les envisageons dans trois directions: (1) Mise au point de techniques permettant la mesure directe du débit circulatoire médullaire (nous y reviendrons au paragraphe consacré à l'ischémie médullaire); (2) Mise au point de tests permettant de juger la tolérance à l'obstruction des afférences antérieures. L'une des possibilités consiste à réaliser une oblitération transitoire de l'artère en cause à l'aide de la sonde d'angiographie. À chaque fois que nous avons effectuée cette manœuvre (au niveau de l'artère d'Adamkiewicz) nous avons observé des réactions péjoratives (secousses cloniques des membres inférieurs avec majoration régressive après levée de l'obstruction du déficit moteur) qui nous ont semblé interdire l'interruption (ligature ou embolisation) de l'afférence; (3) Étude de nouvelles voies d'approche chirurgicale pour tenter d'attaquer directement les artères centrales en respectant l'axe spinal antérieur.

### Angiographie de l'ischémie médullaire

Nous limiterons volontairement notre étude à l'ischémie d'origine athéromateuse dans le territoire de l'artère d'Adamkiewicz dont nous possédons cinq observations avec bilan angiographique concordant.

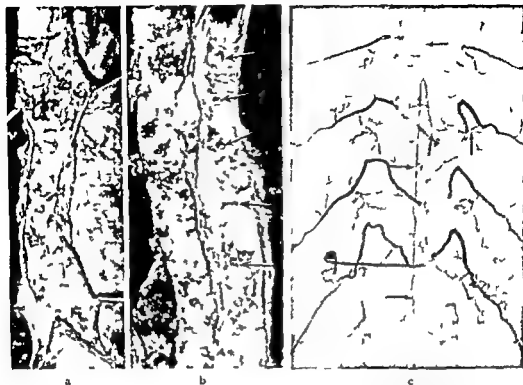


Fig. 7. Athérome aortique (cas autopsique). a) Aorte tapissée de plaques d'athérome obstruant les vasa des artères intercostales et lombaires. b) Aorte après endartérectomie libérant les vasa ( $\rightarrow$ ). c) Artère d'Adamkiewicz injectée par la dixième artère intercostale gauche.

**Anatomie et physiologie.** Les segments médullaires dorso-lombaires sont vascularisés par une artère très largement préférentielle (notion de circulation régionale): *arteria radicularis anterior magna* d'Adamkiewicz, dénommée artère du renflement lombaire par LAZORTHES & GOUAZÉ.

Le fait essentiel demeure le caractère le plus souvent unique de cette artère, qui dans 15 pour cent, des cas seulement (origine haute de la cinquième à la huitième artère intercostale) est suppléée par un apport bas situé. Toute anomalie directe ou indirecte affectant le flux sanguin qui emprunte cette artère revêt d'autant plus de gravité que le territoire desservi est étendu (de D8 au conus en moyenne) et que les possibilités de suppléance sont limitées (LAZORTHES & GOUAZÉ, DI CHIRO et coll.).

En effet, le réseau coronaire péri-médullaire paraît de peu de secours et l'axe spinal antérieur ne constitue pas toujours une voie de suppléance verticale valable (il présente fréquemment une solution de continuité à la jonction des territoires

dorsal superieur et moyen) l'element compensateur le plus efficace est represente par l'anse anastomotique du cone (unissant les deux chaines postero-laterales a la terminaison de l'artere d'Adamkiewicz) a laquelle aboutissent les arteres radiculaires lombo-sacrees anterieures et posterieures. A l'etat normal ces apports inferieurs ne font que « lecher » le cone aux points d'origine des racines mais en cas de deficit des apports arteriels sus-jacents (artere du renflement lombaire) ils sont susceptibles de prendre en charge la vascularisation de la moelle terminale.

**Pathologie.** L'important rapport consacre a la pathologie vasculaire de la moelle par GARCIN et coll (1962) fait une large part aux myelomalaxies par ischémie. Dans l'avant propos de ce rapport GARCIN souligne deux notions essentielles « ce n'est pas tant dans la moelle qu'en dehors d'elle (espace sous-dural, trou de conjugaison massif rachidien, ostium aortique meme) qu'il faut aller chercher les lesions des arteres nourricieres responsables de l'ischémie medullaire a l'instar de ce qui s'est passe pour les ramollissements cerebraux lorsque l'arteriographie nous a revele que certaines thromboses carotidiennes qui en sont responsables se font dans le cou et non dans la boite crânienne » et encore « a cote de la thrombose qui obture (plus rare qu'on ne le croyait) et de la stenose qui retrecit la lumiere des vaisseaux il faut faire une place pour les defaillances circulatoires generales aigues ou les insuffisances circulatoires regionales dans l'etiologie des myelomalaxies ».

Au cours de l'atherome si frequemment rencontre au niveau de l'aorte abdominale chacun de ces deux mecanismes peut jouer de façon isolee ou associee dans la genese d'une insuffisance circulatoire interessante le territoire particulierement expose nous l'avons vu de l'artere d'Adamkiewicz (Fig 7).

**L'angiographie medullaire selective** permet malgre la presence des lesions atheromateuses qui reduisent encore un calibre arteriel deja modeste d'obtenir l'image directe de l'atherome aorto-intercostal et d'objectiver parfois l'ischémie regionale en revelant l'existence d'une circulation de suppléance pathologique. Elle fournit donc un nouveau moyen d'approche diagnostique pour toute une categorie de patients d'age moyen ou mur porteurs d'atteinte medullaire que n'explique aucune etiologie detectable par les procedes d'investigation habituels. En cas de lesions atheromateuses la gene circulatoire est reconnue sur la constatation de deux ordres de signes angiographiques.

**Signes directs.** C'est la stenose ou la thrombose de l'artere (ici l'artere intercostale ou lombaire) qui donne naissance a l'artere du renflement lombaire (Figs 8-9).

L'incidence de face peut montrer une stenose plus ou moins serree de l'artere intercostale ou lombaire dont naît l'artere du renflement lombaire. Stenose qui

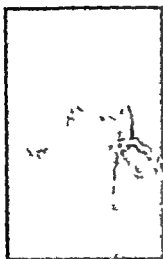
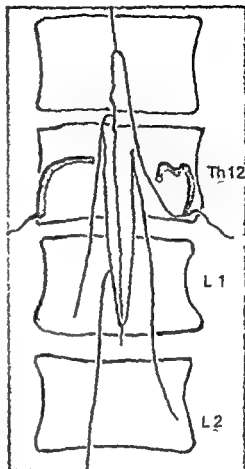


Fig. 8 Angiographie d'une stenose de la deuxième artère intercostale gauche. Face a. La deuxième artère intercostale gauche stenosée n'arrive qu'à l'artère d'Adamkiewicz. L'artère intercostale droite est opacifiée par l'anastomose retro-corporeale et est rectiligne. b. Schéma Opacification du core terminal et des artères radiculaires lombo-sacrées.



« accompagne de sinuosités plus ou moins marquées » contrastant avec la morphologie des artères adjacentes : aspect grêle de l'artère d'Adamkiewicz dont l'opacification est de médiocre qualité. Le rôle d'un « pa. me » artériel surajouté est parfois à retenir.

L'incidence de profil apporte seule un argument de certitude en révélant avec netteté la stenose unique ou multiple siégeant à l'ostium ou sur le premier centimètre de l'artère intercostale ou lombaire responsable. On peut voir sur les clichés se superposer l'artère stenosée et son homologue normale et parfois même on découvre juste en aval du rétrécissement une dilatation post-sténotique très évocatrice.

Pour bien apprécier l'état de l'ostium il est toujours nécessaire de pratiquer une opacification segmentaire de l'aorte en regard ce qui permet d'objectiver

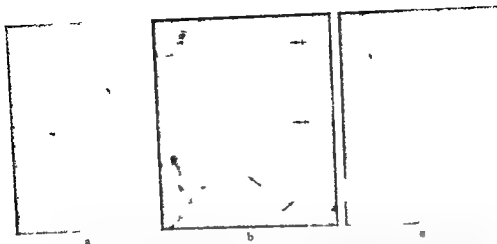


Fig 9 Stenose de la première artère lombaire gauche a) Angiographie de la première artère lombaire gauche stenosee. b) Angiographie de la douzième artère intercostale droite. Revascularisation par les artères musculaires de la première artère lombaire droite. Revascularisation par l'anastomose retro-corporeale (→) de la première artère lombaire gauche opacifiant l'artère d'Adamkiewicz (→→) c) L'origine de l'artère lombaire gauche

la plaque d'athérome qui épaissit la paroi aortique postérieure et empiète plus ou moins sur l'ostium. Il est légitime d'évoquer une obstruction totale et l'ostium lorsque le cathétérisme est impossible alors que l'injection est obtenue grâce au réseau anastomotique musculaire par l'artère intercostale ou lombaire contralatérale. Bien entendu avant d'affirmer l'absence d'injection de l'artère du renflement lombaire par thrombose de sa branche aortique d'origine, toutes les artères intercostales ou lombaires théoriquement susceptibles de lui donner naissance doivent être étudiées par une courte scéno-graphie en se souvenant qu'en cas de stenose ostiale très serrée l'opacification de l'artère d'Adamkiewicz ne peut se faire que très tardivement.

**Signes indirects.** C'est la mise en évidence d'un réseau collatéral de suppléance. Sur l'angiogramme normal de face on observe habituellement l'opacification de l'artère intercostale ou lombaire en amont de la naissance de l'artère d'Adamkiewicz puis cette artère elle-même qui présente un aspect caractéristique avec sa première portion ascendante paramédiane puis sa boucle en « épingle à cheveux » enfin ses deux branches médianes ascendante plus grêle que la descendante qui décrit parfois quelques sinuosités au niveau de sa partie terminale. Sur l'incidence de profil ces différentes composantes sont également retrouvées. L'axe spinal antérieur apparaissant rectiligne quelques millimètres en arrière du rebord vertébral postérieur. Dans l'état actuel des techniques l'angiographie s'avère incapable d'opacifier les artères centrales normales ou a fortiori sténosées.

En l'absence d'insuffisance circulatoire, on n'obtient jamais l'opacification de l'anse anastomotique du cône ni des artères radiculaires lombo-sacrées. Mais lorsqu'existe un défaut d'irrigation régionale (quelle qu'en soit la cause) ce réseau anastomotique peut être retrouvé sur les temps intermédiaires ou tardifs de la sialographie et témoigne alors indirectement de la réalité d'une insuffisance dans cette zone. Il s'agit là d'un argument de grande valeur à l'instar de ce qui est admis à l'échelon de la circulation cérébrale concernant la signification des images de revascularisation (re-injection par l'artère ophtalmique ou l'artère communicante antérieure en cas de thrombose carotidienne; re-injection du réseau sylvien par les anastomoses cérébrale antérieure, sylvienne en cas d'obstruction sylvienne etc.). Toujours sous l'angle de la recherche des suppléances et de leur prise en évidence angiographique, il convient de souligner le rôle possible de l'axe spinal antérieur dont il convient donc de tenter d'apprécier la continuité ou la discontinuité par un bilan angiographique complet qui pourra encore déceler des sténoses étagées des voies d'apport qui peuvent retentir sur les possibilités de compensation du réseau anastomotique musculaire péri-rachidien dont le rôle pensons-nous est loin d'être négligeable. Ainsi pourra-t-on noter la disparition des réseaux anastomotiques musculaires normaux sur un ou plusieurs segments par sténoses multiples des intercostales ou à l'opposé une revascularisation de l'intercostale sténosée par les artères contro-latérales sus- ou sous-jacentes.

### Conclusions

L'angiographie médullaire sélective offre la possibilité d'identifier *in vivo* l'athérome aortico-intercostal et son éventuel retentissement sur la vascularisation médullaire tout particulièrement dans le territoire de l'artère d'Adamkiewicz si fréquemment en cause dans les myélopathies ischémiques. Avec un décalage lié au développement tout récent de l'angiographie médullaire on assiste donc dans ce domaine inaccessible aux méthodes neuro-radiologiques classiques à un progrès substantiel qui logiquement devrait comporter les mêmes éléments positifs que la mise en évidence du rôle des lésions sténosantes proximales des gros troncs artériels à destination céphalique dans la genèse de l'insuffisance circulatoire cérébrale. Ces mêmes remarques peuvent s'appliquer au nouvel essor conditionné dans la détection directe grâce aux procédés angiographiques des sténoses coronariennes, mésentériques et rénales. L'analogie avec l'insuffisance circulatoire cérébrale est frappante: siège des lésions vasculaires hors de la moelle et lésions carotidiennes ou vertébrales extra-craniennes. Rôle des suppléances circulatoires par l'arcade cruciale, l'axe spinal antérieur et les réseaux musculaires péri-rachidiens et revascularisation du système carotidien en cas de lésions

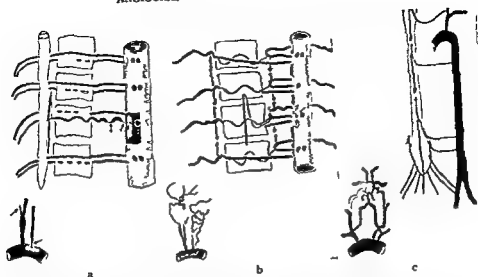


Fig 10 Schemas montrant la similitude de l'ischémie atheromateuse médullaire et cérébrale  
a) Sténose au niveau de l'ostium (→) et au niveau de l'artère d'Adamkiewicz (→) b) Revascularisation de l'artère intercostale sténosée par les anastomoses musculaires entre les artères intercostales homolatérales et contro-latérales (anastomose retro-corporeale) c) Revascularisation de l'artère intercostale sténosée par l'axe artériel spinal antérieur dorsal et cervical et par l'arcade cruciale et les artères radiculaires lombo-sacrées

par l'ophtalmique ou le polygone de Willis. Nécessite d'un bilan circulatoire global intégrant la totalité des apports médullaires et étude des quatre axes artériels dans les atteintes cérébrales.

La signification pathologique des aspects décrits une fois admise leur fidélité à traduire l'existence d'un athérome aorto-intercostal ne doit être retenue qu'en présence d'un contexte clinique cohérent et après élimination d'une autre étiologie par les méthodes conventionnelles (notamment les examens myélographiques la myélographie gazeuse peut du reste apporter des arguments concordants en révélant une atrophie spinale segmentaire). La fréquence de l'athérome à l'âge moyen de la vie autorise en effet toutes les coïncidences.

Un autre point doit être évoqué c'est qu'au niveau de la moelle épinière comme au niveau de l'encéphale l'absence de lésions angiographiquement décelables ne permet pas d'éliminer formellement l'intervention d'un processus circulatoire d'ordre réchémique. C'est à dire que nos préoccupations pour l'avenir doivent tendre vers une précision accrue des investigations et l'on peut dès maintenant prévoir deux orientations dont la réalisation concrète est déjà amorcée au sein de notre équipe : étude de la diffusion des macro-agrégats marqués (albumines iodées) introduits dans la circulation médullaire suivant les voies empruntées par l'angiographie ; détermination du débit circulatoire



medullaire a l'aide de substances marquees (vanon) sur la base de courbes permettant le calcul de la clearance isotopique du produit utilise directement injecte dans l'artere d'Adamkiewicz.

Le dernier probleme concerne les consequences pratiques therapeutiques de ces nouvelles acquisitions. Au dela de leur interet diagnostique, en effet elles invitent a la lumiere des progres recents de la chirurgie vasculaire et de la micro-chirurgie, a reflechir aux modalites techniques d'une amelioration operative des conditions circulatoires pejoratives introduites par l'obstruction progressive d'une artere souvent unique desservant un vaste territoire hautement fonctionnel en l'absence habituelle de suppléance valable. Dans ce debat notre preference va aux solutions actives. L'idée d'une intervention directe sur la jonction aorto intercostale fera nous l'esperons son chemin. Elle implique necessairement un diagnostic precoce avant l'installation de lesions medullaires irreversibles. C'est a souligner qu'en ce domaine comme en matiere de malformations vasculaires spinales, l'angiographie possede des indications specifiques et parfois urgentes si l'on veut pouvoir prolonger la confirmation d'une hypothese diagnostique bien construite par un geste therapeutique efficace.

### **Angiographie medullaire dans le diagnostic et le traitement chirurgical des affections de la moelle epiniere**

La myelographie gazeuse comme les procedes myelographiques utilisant un contraste positif constitue depuis longtemps un moyen sur d'identifier et de localiser les compressions tumorales de la moelle epiniere. Cependant, ces techniques ne fournissent pas toujours les renseignements etiologicals que l'on serait en droit d'attendre d'un bilan pre-operatoire et surtout ne connaissent totalement la composante vasculaire des lesions.

L'angiographie medullaire vient avec bonheur combler cette importante lacune. Notre experience dans ce domaine porte actuellement sur 70 compressions tumorales de la moelle epiniere et de cette experience nous ne retiendrons que les plus importantes.

*Les tumeurs vertebro epidurales.* Pour cette variéte topographique particuliere, l'apport de l'angiographie medullaire est double. Premièrement l'angiographie permet indiscutablement une approche etiologicalue plus precise que celle habituellement fournie par les procedés classiques. Ceci est particulierement vrai en ce qui concerne la distinction entre neoformation vertebrale benigne et neoformation vertebrale maligne. Parmi les tumeurs benignes les angiomes osseux vertebraux dont nous avons etudies jusqu'a maintenant six cas presentent une individualite angiographique certaine. L'aspect angiographique de la vertebre

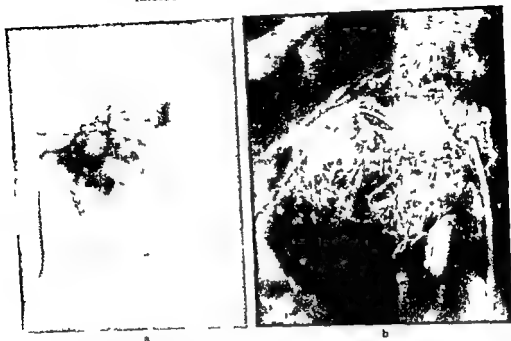


Fig 11 a) Métastase volumineuse opacifiée par la quatrième artère lombaire gauche (fistules artério-veineuses précoces) b) Neurinome en sablier opacifié par la douzième artère intercostale droite (lisière bordant pas de fistules précoces)

angiomateuse est caractéristique, fait de lacunes vasculaires confluentes de volume variable occupant la totalité de la vertèbre et s'étendant aux lames vertébrales. Sur les temps précoces on met souvent en évidence une ou deux artérolas dilatées sinuées injectant la vertèbre pathologique. Sur les temps tardifs l'opacification perd son caractère lacunaire, devient diffuse, dense, homogène et s'étend à la totalité de la vertèbre. L'angiographie permet en outre d'apprécier l'extension extra-osseuse de la tumeur notamment en arrière vers le canal rachidien sur les incidences de profil. Enfin en présence d'une telle étiologie il est très important d'éliminer une malformation vasculaire médullaire associée et ceci seule l'angiographie peut le faire avec sécurité.

Dans les autres variétés histologiques bénignes l'opacification est inconstante.

Par contre lorsqu'il s'agit d'un processus tumoral malin (réticulosarcome ou métastase) l'angiographie permet en règle d'affirmer la nature maligne des lésions sur l'existence aux temps artériolaires de fistules artério-veineuses précoces, sur la mauvaise limitation de l'injection tumorale sans lyse débordante, image assez proche de celle observée dans les néoformations malignes en général. Du



Fig. 1. — Miel néurome dorsal. Angiographie sélective. Opacification hémicène de la tumeur avec artères d'artériennes peritumorales dilatées.

fait de l'injection pathologique l'extension intrarachidienne de la tumeur parfois considérable peut être facilement délimitée sur l'angiographie.

Le second point d'intérêt de l'angiographie réside en l'identification du niveau de pénétration des afférences artérielles médullaires par rapport à la vertèbre pathologique. Ce renseignement se conçoit  $\square$  est précieux lorsqu'est envisagée une intervention chirurgicale mais aussi pour une éventuelle décision de traitement radiothérapique.

Nous pensons que certains accidents chirurgicaux ou post-radiothérapiques sont liés à une lésion vasculaire. L'angiographie permet de les prévoir et de les éviter.

*Les tumeurs extra-médullaires intra-durales (neurinomes, méningiomes).* En ce qui concerne les neurinomes rachidiens nos constatations permettent de penser que dans deux tiers des cas environ l'on obtient angiographiquement une opacification de la tumeur tout à fait analogue à celle observée dans les autres localisations en particulier dans les neurinomes de l'acoustique. La présence de cette



Fig 13 Angiographie vertebrale gauche a) Tumeur intra medullaire opacifiée par l'artère du renflement cervical b) Tumeurs au niveau de C1 et dans la fosse postérieure (→) c) Le pôle inférieur de la tumeur est injecté par la quatrième artère intercostale gauche Trois petites tumeurs dorsales sont aussi injectées (→)

injection pathologique est précieuse pour juger de l'extension des tumeurs qui comportent un prolongement extra rachydien (neurinome en sablier Fig 11 b) Nous ne possédons qu'une seule observation de méningiome dans lequel l'angiographie a permis d'obtenir une opacification tumorale (Fig 12)



Fig. 14. Tumeur intra-médullaire non injectée (astrocytome kystique). La tumeur refoule la branche descendante de l'artère d'Adamkiewicz naissant de la douzième artère intercostale gauche.

Comme dans la catégorie tumorale précédente l'angiographie présente ici encore un énorme intérêt de l'identification des afférences artérielles médullaires normales. Ce renseignement est évidemment capital pour le chirurgien qui pourra mener son exérèse en toute sécurité du fait de la connaissance préalable des rapports dangereux et notamment des rapports vasculaires de la moelle.

*Tumeurs intra-médullaires.* Nous pouvons faire état de six néoformations d'origine gliale (3 ependymomes, 1 astrocytome, 2 gliomes kystiques) et de trois observations concernant les hémangioblastomes intra-médullaires. En ce qui concerne les tumeurs d'origine gliale, il est rare d'obtenir une opacification pathologique. Cet élément négatif apporte une précision sur l'étiologie de la néoformation et n'est pas indifférent à la démarche chirurgicale.

En dehors de toute néovascularisation, il est parfois possible de retrouver sur les angiographies un reflet indirect de l'augmentation de volume de la moelle par l'écartement des axes spinaux postérieurs qui en marque le contour et aussi une déformation de l'axe spinal antérieur dévié latéralement sur les incidences de face ou refoulé vers l'avant sur les incidences de profil.

En matière de tumeurs vasculaires intra-médullaires, c'est-à-dire d'hémangio-

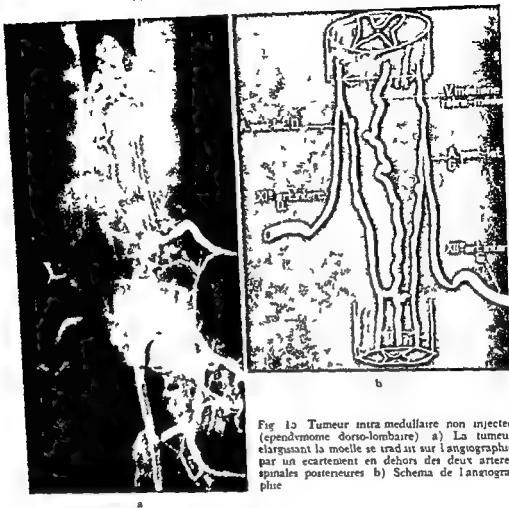


Fig 13 Tumeur intra medullaire non injectee (ependymome dorso-lombaire) a) La tumeur elargissant la moelle se traduit sur l'angiographie par un ecartement en dehors des deux arteres spinales posteneures b) Schema de l'angiographie

blastomes l'angiographie constitue l'examen indispensable au diagnostic comme au traitement

L'aspect angiographique de ces tumeurs vasculaires est tres caracteristique et entierement superposable a celui observe dans les hemangioblastomes cerebel leux L'opacification de ces tumeurs est constante et se fait sous forme d'un fin lacs de faisceaux entremeles associe a des flaqes vasculaires denses ethomogenes qui traduisent angiographiquement la structure histologique de ces neoformations (Fig 13)

L'etude angiographique permet aux temps arteriels un bilan tres precis des pedicules nourriciers de la tumeur dont l'importance chirurgicale est essentielle

Aux temps intermediaires de la serigraphie, l'injection tumorale persiste en

regle pendant 20 à 22 secondes dessine nettement la lésion et permet de juger de son volume et de son siège exact à l'intérieur de la moelle.

Les incidences de profil montrent que le plus souvent les tumeurs prédominent à la partie postérieure de la moelle.

Sur les temps tardifs on étudiera les veines de drainage volontiers volumineuses et qui s'étendent parfois à distance de la tumeur elle-même. L'apparition relativement tardive de ces veines implique une programmation étalée de la sctiographie. Sur le plan purement angiographique trois éléments nous paraissent essentiels pour différencier angiome intra-médullaire et malformation vasculaire. D'une part l'apparition relativement tardive des larges plaques vasculaires tumorales auquel se superpose un larcis vasculaire plus finement injecté, d'autre part la netteté de l'opacification tumorale et sa persistance. En troisième lieu l'injection tardive du réseau veineux de drainage. Ces trois éléments s'opposent à la succession rapide des images artérielles artérioveineuses et veineuses observées dans les angiomes ou l'importance des shunts artérioveineux qui définissent l'affection conditionnent une accélération circulatoire caractéristique.

À côté de son intérêt diagnostique l'angiographie médullaire et elle seule, permet de façon précise un bilan complet des lésions. Notre expérience permet de confirmer les constatations classiques insiste sur la fréquence de localisations multiples aboutissant à la notion de maladie dysembryogénétique c'est le cas de Von Hippel-Lindau. Sept de nos 13 observations comportant sur le seul plan médullaire des lésions associées. Chez l'un de nos malades nous avons pu dénombrer jusqu'à 5 tumeurs médullaires.

En dehors même des difficultés thérapeutiques tirées de l'association à des localisations rachidiennes ou cérébelleuses l'angiographie apparaît ainsi comme le préalable indispensable pour toute discussion de l'indication opératoire chez ces patients.

Lorsqu'il apparaît possible ou raisonnable d'aborder chirurgicalement ces tumeurs vasculaires intra-médullaires la connaissance préalable exacte de la structure anatomique de la tumeur et de son mode de vascularisation tels qu'ils apparaissent à l'angiographie représentent une garantie supplémentaire de succès pour le chirurgien.

### Conclusion

Dans notre esprit l'angiographie médullaire ne doit pas être opposée aux procédés neuroradiologiques classiquement utilisés pour l'identification des compressions tumorales de la moelle épinière. Elle ne saurait prétendre se substituer aux méthodes angiographiques elle les complète souvent très utilement et il arrive qu'elle les dépasse en précision.

# RÉSUMÉ

L'angiographie médullaire est essentiellement utilisée pour l'identification et l'approche thérapeutique des malformations de la moelle. L'étude des malformations médullaires portant sur près de 80 cas a permis un progrès considérable dans le diagnostic radiologique et dans le traitement chirurgical de la maladie. Soixante-dix cas ont été étudiés apportant des renseignements s'ajoutant en qualité à ceux de la myélographie. Dans les médullopathies ischémiques l'angiographie seule peut apporter des arguments diagnostiques.

# SUMMARY

Angiography of the spinal cord serves to identify abnormalities and to decide the lines of treatment. This examination of malformations in nearly 80 cases has led to considerable progress in their diagnosis and helped to plan surgical treatment more efficiently. In 70 of the cases useful information additional to the myelographic findings was gained. Angiography alone is of diagnostic value in ischaemic conditions of the spinal cord.

# ZUSAMMENFASSUNG

Die Angiographie des Rückenmarkes ist eine diagnostische und therapeutische Untersuchungsmethode um Missbildungen zu entdecken. Die angiographischen Erfahrungen an beinahe 80 Fällen von Missbildungen des Rückenmarks führten zu Fortschritten in einer genaueren Diagnose und besseren Planung in der Chirurgie. In 70 der untersuchten Fälle war es möglich die myelographischen Befunde wertvoll zu ergänzen. In Fällen von ischämischen Erkrankungen ist allein die Angiographie von Nutzen.

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Fig 2 Cas 2 Angiographie carotidienne primitive gauche de profil Hypertrophie de la partie initiale de l'artère ophtalmique d'où naît la branche antérieure de la meningée moyenne hypertrophiée qui se termine dans une malformation sagittale

On voyait que là encore la malformation était opacifiée par l'intermédiaire de branches de la maxillaire interne et de la temporale superficielle (Fig 1c)

L'étude radiologique de cette malformation s'était donc trouvée fortuitement réalisée de façon très complète. Elle nous a permis grâce à la soustraction de mieux différencier les pédicules et ce cas est une illustration de l'importance des anastomoses non seulement entre les branches des artères ophtalmiques mais aussi entre les branches de celle-ci et de l'artère maxillaire interne.

L'intervention pratiquée montrait un hématome sous dural en voie d'organisation épais de 1 cm à maximum frontal mais s'étendant très loin en arrière sur la convexité. On reclinait facilement le lobe frontal et on voyait la malformation qui avait le volume d'une cerise et était enchâtonnée dans le lobe frontal au ras de la ligne médiane. À son pôle supérieur une veine artérialisée unique de volume modéré rejoignait la dure mère de la faux près du sinus longitudinal supérieur. À son pôle inférieur un gros tronc d'aspect artériel perforait la dure mère de la lame criblée. On devait interrompre d'abord la veine de drainage pour faire le tour de la face interne de l'ampoule ectasique. Celle-ci était parfaitement libre dans l'espace sous dural et son pédicule artériel avait environ 1 cm et demi de longueur. On plaçait 2 clips sur ce pédicule et on enlevait l'ampoule artérielle en masse.

Les suites opératoires étaient extrêmement simples. L'angiographie de contrôle de l'artère carotide primitive gauche montrait le retour à un calibre normal de l'artère ophtalmique et une morphologie normale de l'artère carotide externe gauche.

On peut donc penser que la malformation était nourrie à la fois par les deux carotides internes et les deux carotides externes.

**Cas 2** Homme âgé de 50 ans présentait un tableau brutal d'hémorragie méningée avec hémiparésie gauche massive au membre inférieur. L'angiographie carotidienne primitive droite montrait une malformation parasagittale fronto-coronale droite drainée par le sinus

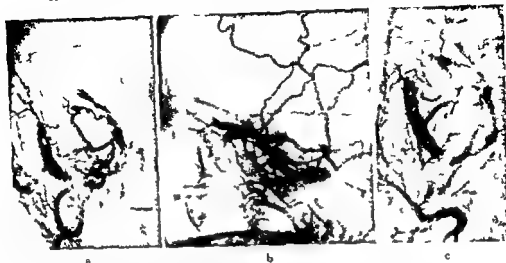


Fig 3 Cas 3 Angiographie de la carotide externe droite Incidence de face haute (a) profil (b) et semi axiale (c) L'artere meningee moyenne très hypertrophiée se termine en se jettant à plein canal dans la veine meningee moyenne elargie de calibre irregulier parallele dans sa première partu à l'artere et les veines de l'orbite en avant et notamment la veine ophtalmique supérieure qui est interrompue dans son 1/3 antérieur (interruption confirmée par une phlebographie orbitaire) et vers les sinus piteux en arrière

longitudinal supérieur et alimente par une volumineuse branche de l'artere ophtalmique (branche antérieure de la meningee moyenne qui naît ici de l'ophtalmique) (Fig 2)

L'intervention pratiquée confirmait cet aspect et montrait que la malformation était située dans l'angle de la convexité et de la faux elle recevait la grosse artere meningee et se drainait dans le sinus longitudinal auquel elle adhérait étroitement. Il existait un hématoxe sous dural et un hématoxe intra cérébral

Il s'agissait là encore d'une malformation artério veineuse élémentaire court-circuitant une branche meningee de l'artere ophtalmique et le sinus longitudinal supérieur

Cas 3 Homme age de 41 ans était admis en novembre 1967 Les premiers troubles remontent à 1960 époque à laquelle le malade aurait ressenti au niveau de l'oreille droite une impression de sifflement qui aurait duré 8 mois pour disparaître ensuite

Au début de 1967 apparaissent des céphalées unilatérales droites et de nouveau du bruit dans l'oreille homolatérale Une vaso dilatation conjonctivale était notée en juin 1967 du même côté Enfin 2 mois plus tard le malade se reveillait avec l'œil droit exorbité et œdématisé Il était alors hospitalisé dans un service d'ophtalmologie où l'on constatait l'existence d'une exophtalmie droite non pulsatile axiale mais variable dans son importance L'acuité visuelle de ce côté était de 10/10e avec correction et l'examen du fond d'œil montrait une hyperhémie papillaire avec turgescence veineuse et quelques hémorragies en flammèches Le reste de l'examen clinique était normal

Le bilan angiographique apportait la solution du diagnostic étiologique de cette exophtalmie unilatérale En effet l'opacification de la carotide primitive puis de la carotide externe permettaient de découvrir une malformation vasculaire de la partie externe de la

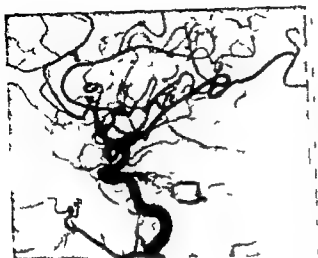


Fig 4 Cas 4 a) Angiographie carotidienne primitive droite de profil. Il existe une malformation vasculaire complexe dont les pedicules semblent provenir surtout de branches de la maxillaire interne a ec opacification precoce du sinus caverneux qui se draine en avant par la veine ophtalmique superieure tres hypertrophiee b) Angiographie carotidienne interne de profil. Il existe au moins trois pedicules naissant du siphon carotidien a la jonction C3—C4 a direction anterieure C3—C5 a direction antero-inferieure et un autre a direction posterieure c) Angiographie carotidienne externe de face. Les pedicules de la malformation proviennent aussi des branches de la maxillaire interne comme le laissent prevoir le cliché a) de l'artere meningee moyenne et de branches directes entre la terminaison de la maxillaire interne et le sinus caverneux passant par le trou grand rond. Ces branches ont ete clippees lors de l'exophtalmie



fosse temporale (Fig 3). Elle etait alimentee par une volumineuse artere meningee moyenne et se vidait dans le sinus caverneux par la veine homologue. Le sinus caverneux se drainait a son tour dans le sinus lateral et dans l'orbite par la veine ophtalmique superieure. Celle-ci paraissait interrompue ju te avant sa terminaison. Cette obstruction etait confirmee par la phlebographie orbitaire.

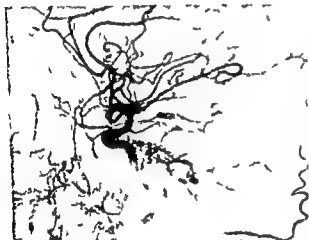
L'intervention pratiquée permettait de decouvrir sur la face externe et inferieure du pole temporal une lesion intradurale allongee et battante. Celle-ci etait en connexion directe en bas en arriere et en dedans avec l'artere meningee moyenne tres dilatee qui etait liee au trou petit rond. La fistule n'etait alors plus battante, mais quand on cherchait apres avoir incise le feuillet externe de la dure mere a la cliver on s'apercevait qu'elle adherait



a



b



c

Fig 5 Cas 5 Angiographie carotidienne primitive gauche de profil temps precoce Malformation angiomateuse vascularisee par des branches du siphon carotidien debut d'opacification dans sa partie postérieure d'une veine de drainage La carotide externe ne semble pas participer a la vascularisation de la malformation ce qui a ete confirme par une angiographie selective de la carotide externe pratiquée secondairement b) Temps tardif La malformation angiomateuse est mieux opacifiée et se draine notamment vers le sinus petreux superieur c) Angiographie carotidienne gauche de face La malformation angiomateuse et sa veine de drainage se pro-

jetten en dehors du siphon carotidien Elle est situee sur le bord libre de la tente du cerveau, ce que confirme encore une angiographie en incidence semi axiale (non representee ici) Les angiographies carotidiennes droites et vertebrales etaient normales

au cortex de la face externe du pole temporal Celui-ci avait d'ailleurs une couleur chamois sequele manifeste d'un saignement ancien Par contre plusieurs arterioles intra-durales etaient clippees en faisant le tour de la fistule et communiquaient vraisemblablement avec elle Quant a la veine menagée efférente qui etait accolée a son artere homologue elle etait sectionnée apres ligature au pole infero-interne de la lesion Ainsi celle-ci pouvait etre enleee en masse

Les suites opératoires étaient extrêmement favorables puisque dans les heures suivantes l'exophtalmie droite avait pratiquement disparu.

L'examen histologique sur des coupes en séries conclut à un aspect typique de fistule intracerveuse.

Le malade a été revu quatre mois plus tard et le problème ophtalmique paraissait totalement réglé.

*Cas 1* Femme âgée de 34 ans sans antécédent particulier perçoit au décours d'un accouchement un souffle imminent temporal droit un an après apparaitrait progressivement une exophtalmie unilatérale droite non pulsatile avec hyperhémie et diplopie qui la font consulter.

L'angiographie carotidienne primitive montre l'existence d'une fistule semblant carotido-cervineuse avec opacification immédiate de la veine ophtalmique supérieure droite très diluée (Fig. 1a).

En fait les angiographies sélectives (Fig. 1b-c) montrent qu'il s'agit d'une malformation dure mérienne complexe avec des multiples pédicules carotidiens externes (méninges moyennes branches dirigées vers la tumeur intracerveuse et sinus cavernaux) et internes (branches inférieures du siphon).

Le drainage veineux se faisait très précocement par le sinus cavernaux vers la veine ophtalmique supérieure droite principalement. L'intervention a permis que de lier les pédicules provenant de la carotide externe ce qui a entraîné une discrète régression de l'exophtalmie.

*Cas 2* Homme âgé de 17 ans originaire du Mali sans antécédent avait une hémorragie méningée sans signe de localisation à l'EEG il existait des ondes lentes temporo-pariétales gauches.

L'angiographie carotidienne primitive gauche montre des artères cérébrales en place mais il semblait exister une opacification précoce du sinus pétreux supérieur. La soustraction permettait de mettre en évidence une malformation anéurysmale vasculaire par des artères méningées du siphon siégeant au niveau du bord libre de la tente du cervelet (Fig. 5).

Il était alors procédé à une angiographie carotidienne externe gauche puis externe et interne droite qui étaient normales de même que l'angiographie vertébrale.

Le malade refusait l'intervention qui lui était proposée.

## Discussion

Ces cinq observations illustrent le polymorphisme des malformations dure mériennes tentorielles et sub-tentorielles.

Si des hémorragies méningées ou des hématomas ou duraux peuvent être révélateurs certaines ont été révélées par une exophtalmie unilatérale quant aux souffles intracrâniens ils paraissent rares dans cette localisation.

Le diagnostic en est essentiellement angiographique et il faut insister sur l'importance d'une étude précise de la vascularisation dure mérienne c'est-à-dire l'intérêt de l'angiographie de la carotide externe dans une exophtalmie unilatérale même si elle n'est pas typiquement vasculaire, dans la recherche étiologique d'une hémorragie méningée voire d'un hématome sous-dural spontané si les angiographies de la carotide interne et de la vertébrale sont normales.

Il faudra penser à ces malformations quand apparaissent précocement des images veineuses, même localisées (Cas 4) et faire alors une soustraction des clichés et des angiographies sélectives externe et interne homo- et contre-latérales les artères menéges des territoires adjacents apparaissent d'ailleurs souvent hypertrophiées même si elles ne participent pas directement à la vascularisation angiomateuse (vascularisation de compensation?)

Il est généralement facile d'éliminer les malformations angiomateuses cérébrales qui peuvent avoir des pédicules menéges, mais qui sont alors souvent très graves.

Quant aux fistules menéges post-traumatiques, notamment carotido-caverneuses la notion de traumatisme ancien ou récent le caractère souvent pulsatile de l'exophtalmie et la découverte d'une fistule directe entre la carotide interne et le sinus caverneux ou une artère et une veine menége, en feront le diagnostic.

### Conclusion

Les malformations vasculaires dure méniennes ne semblent plus avoir de caractère exceptionnel et le problème est de savoir y penser devant une hémorragie menége ou sous durale une exophtalmie ou un souffle leur bilan angiographique devra comporter souvent l'étude de la vertébrale ainsi que des quatre pédicules carotidiens la soustraction des clichés est en général indispensable.

### RÉSUMÉ

Les malformations dure méniennes de la fosse postérieure sont bien connues et leur tableau clinique et angiographique assez stéréotypé. Celles sus-tentorielles sont moins fréquentes et plus polymorphes révélées cliniquement par une hémorragie menége ou sous durale parfois par une exophtalmie et plus rarement un souffle intracranien. L'angiographie de la carotide externe est généralement la plus parlante mais dans les localisations médianes et antérieures c'est l'angiographie carotidienne interne qui permet leur opacification. Dans quelques cas l'existence de pédicules multiples rend nécessaire la réalisation des deux angiographies bilatéralement sous des incidences multiples. La soustraction doit être systématique pour l'analyse des pédicules de la malformation.

### SUMMARY

The dural angiomas of the posterior fossa present a well known clinical and angiographic entity those situated supratentorially are rare and of a more varying type. Clinically these formations are revealed by a meningeal or subdural haemorrhage occasionally through exophthalmos or perhaps even by a cranial bruit. Angiography of the external carotid artery

generally results in characteristic appearances although when lying in the midline and anteriorly these angiomas require angiography of the internal carotid artery. The presence of a multiple stalk like supply may make bilateral angiography with multiphase films necessary. The subtraction method may assist in the complete analysis.

## ZUSAMMENFASSUNG

Die Durarangiome der hinteren Schädelgrube besitzen ein Krankheitsbild das klinisch und angiographisch ziemlich konstant ist. Falls sie jedoch supratentoriell gelegen sind sind sie selten und haben ein wechselndes Bild. Die ersten klinischen Zeichen einer meningale oder subdurale Blutung ist oder ein Exophthalmus oder in seltenen Fällen ein Geräusch im Schädel. Im allgemeinen ergibt die Angiographie der Carotis externa ein typisches Bild aber falls die Angiome vorne und in der Mittellinie lokalisiert sind muss man die Carotis interna injizieren. Gelegentlich falls stielartige Zufuhrarterien vorhanden sind ist beidseitige Injektion und Aufnahme in mehreren Ebenen erforderlich. Es muss dann auch die Subtraktionsmethode angewandt werden um alle Stielarterien zu erkennen.

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## VEINS OF THE UPPER CERVICAL SPINAL CORD IN VERTEBRAL ANGIOGRAPHY

by

T O GABRIELSEN J F SEEGER and J D CRANE

SCHUCHTER & ZINGESSER (1966) have stated that lateral roentgenograms of selective vertebral angiography demonstrate the anterior spinal artery fairly frequently and the posterior spinal arteries rather infrequently. In our experience veins outlining the anterior and posterior borders of the upper part of the normal or displaced cervical spinal cord can also be demonstrated.

*Anatomy.* In an investigation of cervical spinal cords from cadavers TURNBULL et coll (1966) found that the vein accompanying the anterior spinal artery lay deep to the artery in the anterior median fissure. At times it came out onto the surface a little lateral to the fissure. Often a single large vein ran the length of the cervical cord on its posterior surface but in many specimens there were 2 posterior spinal veins or an ill defined plexus. The veins on the surface of the cord were in communication with one another and with radicular veins (and thus the paraspinal veins).

The spinal cord veins are also continuous with the veins of the posterior cranial fossa (DJINDJIAN et coll 1970 DI CHIRO & DOPPMAN 1970).

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During part of this investigation J F Seeger was a NINDS Special Fellow No 1 F11 NS07365-01



*Material and methods* Fifty recent, successive, routine vertebral angiographies were reviewed without election regarding diagnosis. Only selective vertebral angiographies showing at least the atlas and axis in lateral views were included. The age distribution of the patients, 30 males and 20 females, was as follows: 2 patients between 0 and 9 years, 3 between 10 and 19, 8 between 20 and 29, 9 between 30 and 39, 13 between 40 and 49, 13 between 50 and 59, 1 between 60 and 69, and 1 between 70 and 79 years. There were 34 left and 16 right vertebral angiographies, all performed by catheterization from the femoral artery without aid of general anesthesia. A 5 ml dose of Conray (meglumine iothalamate 60 %) was usually injected either by hand or pressure injector over a one second period. Lateral and half axial a.p. roentgenograms were obtained by alternate exposures on Elema Schonander biplane cut film changers AOT 10/12 using a 40 inch (1 m) target film distance. Subtraction was always attempted.

### Results and Discussion

HUANG & WOLF (1970) have indicated in drawings of the posterior cranial fossa veins that the veins of the brain stem are in direct continuity with those of the cervical spinal cord. DI CHIRO & DOPPMAN (1969) published a case of hemangioblastoma of the spinal cord with contrast filling of cervical cord veins at angiography. The same authors (1970) as well as DJINDJIAN *et coll.* (1970) have recently called attention to the fact that angiography of spinal cord angiomas may demonstrate huge spinal veins with partial drainage intracranially. The last authors maintain that conventional methods of angiography in the healthy subject fail to reveal the spinal veins except on rare occasions. GABRIELSEN & AMUNDSEN (1969) incidentally pointed out the cervical cord veins in an illustration of vertebral angiography performed on a child with cerebral aqueduct stenosis (Fig. 1). With this possible exception we have found no previous reference which has demonstrated the normal spinal cord veins by means of angiography in living humans.

The frequency and relative degree of demonstration of the arteries and veins of the upper cervical spinal cord in the present material are given in the Table. The arteries are included in this tabulation primarily in order to examine their appearance and incidence of contrast filling compared with the corresponding veins for localization of the cervical spinal cord. Such a comparison also serves as an indication of the technical quality required to demonstrate the spinal cord veins by vertebral angiography.

SCHIECHTER & ZINGESSER (1966) identified the anterior spinal artery in 50 % and the posterior spinal plexus in 4 % of lateral roentgenograms from vertebral



Fig 1



Fig 2

Fig 1 Subtraction lateral view of selective right vertebral angiography in 6-month-old boy with cerebral aqueduct stenosis demonstrated by encephalography and ventriculography. The spinal cord and anterior border of pons and medulla oblongata are outlined by veins (arrows). (Reproduced with permission of Radiology.)

Fig 2 Subtraction lateral view of selective left vertebral angiography in 3 year old boy with severe cranio cerebral trauma without significant localized intracranial expansion. Excellent demonstration of spinal cord veins (arrows) by subtraction in spite of patient motion.

angiographies performed by direct puncture of the vertebral artery. This is approximately the frequency of arterial demonstration rated as good in our series. There was, in addition, faint filling of the anterior spinal artery and posterior spinal arteries in 44 % and 28 % of our cases respectively (see Table). These discrepancies can readily be explained on the basis of recent technical advances and perhaps by differences of opinion in regard to whether a very faintly filled artery can or cannot be reliably identified.

Vertebral angiographies of excellent technical quality are necessary to demonstrate the upper cervical cord veins in most patients. We could usually identify

Table

*Frequency of demonstration of arteries and veins of the upper cervical spinal cord in the present material*

	Degree of demonstration			
	None	Faint	Good	Faint or good
<b>Lateral film</b>				
Anterior arteries	3 (6 )	22 (44 )	25 (50 )	47 (94 )
Posterior arteries	31 (62 )	14 (28 )	5 (10 )	19 (38 )
Anterior veins	14 (28 )	16 (32 )	20 (40 )	36 (72 )
Posterior veins	27 (54 )	17 (34 )	8 (16 )	23 (46 )
<b>Frontal film</b>				
Arteries	25 (50 )	18 (36 )	7 (14 )	25 (50 )
Veins	46 (92 )	3 (6 )	1 (2 )	4 (8 )

the anterior upper border of the spinal cord better during the arterial than venous phase whereas the reverse was true for the posterior border (see Table). There was either good or faint filling of the anterior cord veins in 72 % and of the posterior cord veins in 46 % of the cases in lateral view. Faintly filled vessels were ordinarily seen over only a short segment of spinal cord. It sometimes required careful correlation between the arterial and venous phase to be certain that faintly filled vascular structures really were demonstrated. Even faint filling of arteries and veins was then helpful in localizing the spinal cord.

The spinal veins can seldom be reliably identified in a p views of vertebral angiographies (see Table). However we have on rare occasions seen the lateral borders of the cervical spinal cord clearly outlined by veins in infants using subtraction. Subtraction is quite helpful in the demonstration of the anterior and posterior spinal veins, even when the patient has moved somewhat (Fig 2).

Use of general anesthesia would probably provide better subtraction and consequently better demonstration of spinal cord veins by decreasing patient motion. Magnification techniques may also prove valuable. Improved angiographic demonstration of spinal veins would undoubtedly result if the dose of contrast medium were increased. More than 5 ml might be delivered safely in the vertebral artery in many patients particularly if care were taken to withdraw the catheter from the artery immediately after the injection.

In our experience reflux of contrast medium into the contralateral vertebral artery will take place more frequently when the catheter tip is placed quite high rather than low in the vertebral artery. We do this with great caution since this reflux phenomenon probably is related to a mild mechanical obstruction by the catheter permitting some elevation of intraluminal pressure in the ipsilateral vertebral artery during contrast injection.



Fig 3



Fig 4

Fig 3 Lateral view of percutaneous transfemoral retrograde right jugular phlebography in 53 year-old man with occlusion of right sigmoid and superior petrosal sinuses due to meningioma and previous operation. Retrograde filling of spinal cord veins (arrows) among numerous other veins.

Fig 4 Subtraction lateral view of selective right vertebral angiography in 33 year-old man with large hemangioma of left side of face. Labeled are veins along posterior border of spinal dura mater (posterior row of arrows), residual contrast medium in dependent part of vertebral artery (anterior row of arrows), faintly shown spinal cord (crossed arrows) and a spinal radicular or paraspinal vein (double crossed arrows).

There was reflux of contrast medium into the opposite vertebral artery in 50% of our examinations usually with associated demonstration of both posterior inferior cerebellar arteries. The veins of the upper cervical spinal cord were seen somewhat more frequently when the catheter tip was located above rather than below the C3—C4 level. We always place the catheter tip farther caudally when evaluating the lower cervical cord by angiography.

The current vertebral angiographies had been carried out for a multitude of clinical reasons. Although the clinical and roentgenographic diagnoses were not tabulated, no obvious relationship between demonstration of spinal cord veins and clinical states such as raised intracranial pressure was observed.

Tableau 2

*Toxicités comparées chez la souris du Dimer X avec le Methiodal et le Contrix 28 injecté par voie intraveineuse. La vitesse d'injection a été de 0,1 ml/min pour le Methiodal et de 2 ml/min pour le Contrix 28 et le Dimer X.*

Solutions	DL	
	Sel g/kg	Iode g/kg
Methiodal	9	47
Contrix 28	11,5	54
Dimer X	13,2	62

Tableau 3

*Neurotoxicité chez la souris du Methiodal, du Contrix 28 et du Dimer X injecté dans les espaces méninges.*

Solutions	DL	
	Sel mg/kg	Iode mg/kg
Methiodal	235	122
Contrix 28	370	150
Dimer X	617	290

Le gain en diminution de la neurotoxicité du Dimer X injecté dans les espaces méninges est considérable puisque la DL<sub>50</sub> est presque deux fois plus élevée par rapport au Contrix 28 et presque trois fois par rapport au Methiodal (Tableau 3).

L'analyse comparative des effets secondaires entre Contrix 28 et Dimer X, (Tableau 4) permet les constatations suivantes, compte tenu du nombre d'examenés un peu moins élevé au Dimer X qu'au Contrix 28. (1) Le nombre des céphalées est sensiblement comparable alors que les autres symptômes sont un peu plus nombreux avec le Dimer X. (2) L'étude des signes radiculaires et irritatifs confirme la moindre neurotoxicité du Dimer X puisque le nombre d'accentuation d'algies pré-existantes et surtout, des crises cloniques est en nette régression par rapport au Contrix 28. Il faut cependant souligner leur persistance qui prouve que l'agressivité de cette drogue pour le système nerveux ne peut pas être négligée. D'ailleurs l'existence d'une crise convulsive dans notre matériel montre bien que si la moindre toxicité de ce produit pour la moelle et les racines semble démontrée, sa diffusion à l'étage cortical peut être à l'origine d'accidents sérieux.

Tableau 4

*Analyse comparative des effets secondaires entre Contrix 28 (847 cas) et Dimer \ (757 cas)*

Effets secondaires	Contrix 28	Dimer \
Signes méninges		
Céphalées	107	100
Vomissements	6	12
Température	4	7
Raideur de la nuque	6	14
Signes radiculaires		
Accentuation des algies pré existantes	47	14
Syndrome de la queue de cheval	1	0
Signes irritatifs		
Crises d'épilepsie	0	1
Crises cloniques	29	7
Lipothymies	0	7
Décès	1	0

Les conditions techniques doivent être très strictes et comparables à celles préconisées pour le Contrix 28 c'est à dire

(1) Ne pas laisser le malade totalement à jeun et éviter toute médication hypotensive

(2) Lieu d'injection L3—L4 ou L4—L5, en position assise verticale. Le contrôle télévisé de l'injection est préconisé pour que le niveau de contraste ne dépasse pas le disque L2—L3

(3) La quantité optimale de Dimer \ est 5 ml pur ou dilué dans la même quantité de liquide céphalo-rachidien ou d'eau distillée soit au total 10 ml maximum. Certains auteurs conseillent le retrait du contraste après l'examen

(4) Position du malade après l'examen. Le maintien en position assise prolongée pendant six à huit heures semble moins essentiel qu'avec le Contrix 28. Cependant pour diminuer les céphalées favorisées par la position assise prolongée nous conseillons de mettre le malade en position déclive, tête relevée pendant quatre à six heures

(5) Prévention des accidents. La surveillance clinique du malade après l'examen est très importante. Si l'on constate une hyperreflectivité tendineuse il est nécessaire de pratiquer une injection intramusculaire de 10 mg de Diazepam (Valium) quatre heures après l'examen

(6) Traitement des accidents. Si des secousses cloniques se déclenchent et si l'injection intramusculaire de 10 mg de Diazepam est inefficace, il est indiqué de faire une injection intraveineuse de 10 mg de Diazepam mais sous le contrôle

permanent d'un anesthésiste réanimateur de façon à pouvoir pratiquer immédiatement une intubation trachéale en cas de dépression respiratoire.

(7) Utilisation du Dimer X pour l'exploration de la moelle dorsale et cervicale. L'expérience actuelle ne permet pas de l'autoriser. En effet, les phénomènes d'irritation médullaire ou cérébrale observés paraissent en rapport avec une concentration trop élevée du contraste dans le liquide céphalo rachidien lors de sa diffusion dans les espaces sous arachnoïdiens. Il est donc nécessaire, pour l'éviter, de ne pas dépasser la dose optimale de 5 ml suffisante pour l'exploration des racines. Or à cette faible concentration le contraste d'une myélographie dorsale ou cervicale est insuffisant pour que l'exploration soit radiologiquement valable aussi bien en radiographie qu'en radioscopie télévisée lors du basculement du malade. La myélographie est donc contre indiquée dans les conditions d'utilisation actuelles du Dimer X.

### Conclusion

Si l'agressivité du Dimer X, pour le système nerveux, est beaucoup plus faible, comme le prouve la régression très nette des crises de contracture clonique, celles-ci n'en demeurent pas moins une complication possible de l'examen.

Il est donc nécessaire d'appliquer rigoureusement les conditions techniques que nous préconisons qui seules, permettront de les éviter.

Ce nouveau produit constitue certainement un progrès dans l'agressivité chimique des produits iodés hydrosolubles résorbables utilisés en radiculographie, mais les dangers que présente son utilisation ne sont pas négligeables.

Nous demandons à ce que cet examen ne soit pratiqué que par des neuro-radiologues, qui connaissent parfaitement aussi bien les techniques de l'examen que ses limites et ses dangers et qu'il ne soit jamais exécuté en dehors d'un milieu hospitalier neuro-chirurgical.

### RÉSUMÉ

Les auteurs présentent les résultats qu'ils ont obtenus en radiculographie lombo-sacrée avec le Dimer X sans rachianesthésie.

### SUMMARY

The results obtained with lumbar myelography with Dimer X without spinal anaesthesia are described.

### ZUSAMMENFASSUNG

Die Ergebnisse der lumbalen Myelographie mit Dimer X ohne Lumbalanästhesie werden beschrieben.

## SPINALF DIAGNOSTIK MITTELS ANGIOGRAPHISCHER METHODEN UNTER BESONDERER BERÜCKSICHTIGUNG DES ELEKTRONISCHEN SUBTRAKTIONSVERFAHRENS

VON

E. LOHR H. E. CLAR und W. BETTAG

Die Diagnostik spinaler Prozesse hat durch die Einführung mehrerer neuer Untersuchungsmethoden an Bedeutung erheblich gewonnen nicht zuletzt deswegen weil die ■ dazu beigetragen haben in vielen Fällen einen gezielten und erfolgreichen neuro-chirurgischen Eingriff zu gewährleisten

Wenn auch die Myelographie bei der Diagnostik spinaler Erkrankungen nicht mehr fortzudenken ist so sind die Untersuchungsergebnisse dieser Methode hinsichtlich der Natur eines Passage stops oder einer Passagebehinderung nicht immer überzeugend Ein besonderes Problem stellen die spinalen Prozesse ohne einen Kontrastmittelstop dar (z B subarachnoidale Blutungen, flache Angiome), die bei der myelographischen Untersuchung häufig keine oder nur geringe pathologische Zeichen aufweisen

Als ein wesentlicher Fortschritt bei der Lokalisation derartiger Erkrankungen sind die angiographischen Untersuchungen des Spinalkanals zu nennen (FISCHGOLD et coll DJINDJIAN et coll DI CHIRO et coll) Hier ist auch die spinale O.-o-Phlebographie zu nennen die insbesondere von GREITZ et coll sowie von



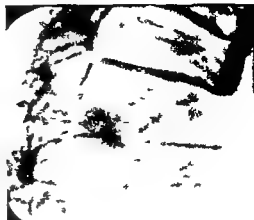


Abb. 1. Spinale Osso Phlebographie. Gefäßreicher extramedullärer Tumor (Angiom) mit Übergreifen auf die Wirbelbogen und Wirbelkörper. Aufnahme in elektronischer Subtraktions- und Vergrößerungstechnik.

VOGELSAANG zu einer wichtigen Untersuchungsmethode ausgebaut wurden. Beide Verfahren eignen sich besonders zur Darstellung von gefäßreichen intra- und extraduralen Prozessen.

Wir mochten über unsere eigenen Erfahrungen an Patienten berichten, bei denen wir teils eine spinale Osso Phlebographie, eine indirekte spinale Angiographie oder beide Untersuchungen kombiniert durchgeführt haben. Im Gegensatz zu dem selektiven Verfahren nach DJINDJIAN et coll. sind wir bei der arteriellen Darstellung von einer Übersichts Aortographie nach HETTLER ausgegangen, wobei der Patient seitlich gelagert wurde. In dieser Projektion erfolgten Serienaufnahmen, welche mittels eines elektronischen Subtraktions- und Harmonisierungsverfahrens unter Verwendung einer elektronischen optischen Vergrößerungstechnik ausgewertet wurden. (Wir verwendeten das elektronische Subtraktions- und Harmonisierungs-Gerät der Firma Siemens, Erlangen.)

In unserem Krankengut fanden wir 3 angiomatöse Tumoren des Rückenmarkes und ein Wirbelkörper Angiom. In 2 Fällen konnten gefäßarme Prozesse einschließlich eines Rezidives eines Meningiomes lokalisiert werden. Die präoperativ gestellten Verdachtsdiagnosen konnten intraoperativ bestätigt werden. Bei 2 Patienten führte eine extramedulläre Veränderung (Bandscheibenprolaps bzw. Bandscheibendestruktion) weder bei der spinalen Angiographie noch bei der Osso Phlebographie zu einem eindeutig pathologischen Befund.

Beim ersten dargestellten Fall (Abb. 1) wurde eine spinale Osso Phlebographie wegen des Verdachtes eines spinalen Tumors durchgeführt. Es zeigt sich ein gefäßreicher extramedullärer Tumor mit Übergreifen auf die Wirbelbogen und Wirbelkörper. Diese Aufnahme in elektronisch-optischer Vergrößerungstechnik läßt die Ausdehnung des gefäßreichen Bezirkes des Angiomes besonders gut sichtbar werden.



Abb 2 Indirekte spinale Angiographie a) Intramedullares spinales Angiom (durch Operation bestätigt) b) Elektronische Harmonisierung Positive Kontrastdarstellung des Rückenmarkes In Höhe der Abdrängung des Pantopaque Kontrastmittels gehen die Konturen des Angiomes teilweise durch die Überlagerung des durch Kontrastmittel angefärbten Rückenmarkes verloren

Bei dem nächsten Fall (Abb 2 a) wurde eine indirekte spinale Angiographie durchgeführt. Es stellt sich ein intramedullär gelegener gefäßreicher Tumor (Angiom) dar, der hinsichtlich seines Sitzes und Ausdehnung durch die Operation bestätigt wurde. Die folgende Abb 2 b zeigt eine Aufnahme derselben Untersuchungsserie unter Verwendung des elektronischen Harmonisierungsverfahrens. Es kommt zu einer positiven Kontrastmitteldarstellung des Rückenmarkes über eine Strecke von mehreren Wirbelkörpern. In Höhe der Abdrängung des Pantopaque Kontrastmittels läßt sich der gefäßreiche Bezirk nur noch angedeutet abgrenzen. Er wird teilweise durch das insgesamt durch das Kontrastmittel sich anfärbende Rückenmark überdeckt.

Im folgenden Fall (Abb 3) bestand klinisch der Verdacht eines Meningiom Rezidivs. Bei der indirekten spinalen Angiographie läßt sich in der seitlichen Projektion in Höhe des sich darstellenden Metallclips der von einer früheren Operation herrührt, deutlich eine Einziehung des Rückenmarkes erkennen. Hier grenzt sich ein kirschengroßer Rundherd ab, der intraoperativ dem Meningiom Rezidiv entsprach.

Bei einer 26-jährigen Patientin, die mehrfach zur Zeit ihrer Periode ein querschnittartiges Syndrom aufwies, haben wir bei der Angiographie in Höhe von Th11—Th12 einen gefäßreichen, offensichtlich intraspinal gelegenen Bezirk entdeckt, der wahrscheinlich auch entsprechend dem klinischen Befund einer Endometriosis externa entsprechen dürfte (Abb 4). Die Patientin wurde bisher noch nicht operiert und befindet sich in weiterer Beobachtung, nachdem zunächst eine hormonelle Therapie eingeleitet wurde.

Wenn wir zur Zeit auch noch nicht über eigene Erfahrungen mit der elektronischen spinalen Angiographie verfügen und unser Untersuchungsmaterial noch relativ klein ist, so sind wir doch der Meinung, daß zur Abklärung von spinalen Prozessen, insbesondere auch von Angiomen, in vielen Fällen die indirekte spinale Angiographie unter Verwendung des elektronischen Subtraktionsverfahrens aus-



Abb 3



Abb 4

Abb 3 Indirekte spinale Angiographie Kurz proximal des Metallelips (von einer früheren Operation herstammend) angedeutete Abgrenzung eines Rundherdes von etwa Hirschengroße der einem Meningiom Reizidus entsprach

Abb 4 Indirekte spinale Angiographie Ein diffuser schlecht abgrenzbarer wahrscheinlich intraspinal gelegener Prozess in der arteriellen Phase der einer Endometriosis externa entsprechen dürfte

reichend ist wobei wir uns über die Grenzen der Leistungsfähigkeit dieser Methode bewußt sind. Das elektronische Harmonisierungsverfahren bietet in der Weise Vorteile, daß hierdurch häufiger das Kontrastmittel angefarbte Rückenmark in größerer Ausdehnung zur Abbildung gelangt. Es besteht der Eindruck, daß dieses Verfahren bei der Angiographie gefäßarmer Prozesse vom Vorteil ist.

Die Osso Phlebographie sollte insbesondere auch nach Ansicht von GREITZ et coll. sowie VOGELSANG bei der Lokalisation von extraduralen vertebrealen und paravertebralen Prozessen zur Anwendung gelangen, insbesondere dann wenn es sich um gefäßreiche Veränderungen (Angiome, Metastasen, Osteomyelitiden) der Wirbelkörper handelt.

## ZUSAMMENFASSUNG

Anhand des eigenen Krankengutes wird über die Ergebnisse der spinalen Osso Phlebographie und der semiselektiven spinalen Angiographie unter Verwendung des elektronischen Subtraktions- und Harmonisierungsverfahrens berichtet. Die Osso Phlebographie eignet sich besonders zur Lokalisation von gefäßreichen extraduralen Prozessen, insbesondere auch von solchen, die auf den Wirbel übergreifen. Die semiselektive spinale Angiographie im Rahmen einer Aortographie erweist sich vorteilhaft bei der Darstellung gefäßreicher Rückenmarkstumoren, insbesondere von Angiomen. Gefäßarme Tumoren können häufig mit dem elektronischen Harmonisierungsverfahren durch Anhebung des Kontrastes besser dargestellt werden.

## SUMMARY

The authors report their results after spinal ossophlebography and semiselective spinal angiography aided by electronic subtraction and harmonisation. Ossophlebography is highly suitable for the localisation of strongly vascular extradural processes particularly when they affect the vertebrae. Semiselective spinal angiography a modified aortography is advantageous in cases of vascular cord tumours such as angiomas. Electronic harmonisation with increased contrast is of great help when dealing with tumours of poor vascularisation.

## RÉSUMÉ

Les auteurs présentent leurs résultats de phlebographie osseuse rachidienne et d'angiographie médullaire semi sélective traitées par soustraction et harmonisation électronique. La phlebographie osseuse convient particulièrement bien à la localisation des lésions extra durales très vasculaires en particulier quand elles atteignent les vertèbres. L'angiographie médullaire semi sélective au cours d'une aortographie est utile pour mettre en évidence les tumeurs médullaires vascularisées en particulier les angiomes. Souvent l'augmentation du contraste grâce à l'harmonisation électronique permet de mieux mettre en évidence des tumeurs peu vascularisées.

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## EXPLORATION ANGIOGRAPHIQUE DES ANGIOMES VERTEBRAUX

par

C MANELFE et R DJINDJIAN

La pratique de l'angiographie medullaire selective par catheterisme femoral retrograde nous a amenes a nous interesser a la vascularisation normale et pathologique de la colonne vertebrale

Nous rapportons ici onze cas d'angiomes vertebraux explores par catheterisme selectif

### Anatomie

L'etude anatomo-radiologique de la vascularisation des corps vertebraux a fait l'objet de nombreux travaux parmi lesquels nous citerons ceux de HIRTL (1873), de WAGONER & PENDERGRASS (1932) et de MARKHASHOV (1965)

*La colonne vertebrale* est vascularisee par des arteres metamériques dont les origines sont differentes suivant les niveaux

Dans la region cervicale (Tableau 1) la vascularisation depend de la disposition topographique des arteres (1) deux sources ont pratiquement constantes les arteres vertebrales et cervicales profondes et (2) trois ont accessoires

Tableau 1

Sources artérielles des vertèbres cervicales (d'après MARKHASTOV) ● Constantes ○ inconstantes

	C1	C2	C3	C4	C5	C6	C7
Vertébrale	●	●	●	●	●	●	○
Cervicale profonde	○	○	●	●	●	●	●
Cervicale ascendante	○	○	○	●	●	○	
Tronc thyro-bicervico-scapulaire						○	○
Thyroïdienne inférieure					○	○	○

Tableau 2

Sources artérielles des vertèbres lombaires et sacrées (d'après MARKHASTOV) ● Constantes ○ inconstantes

	L1 à L3 L4	L5	S1	S2	S3	S4	S5
1ère à 3ème paires art. lombaires	●						
4ème paire art. lombaires	●						
5ème paire art. lombaires		○					
Sacrée moyenne	○	●	●	○	○	○	○
Ilio lombaire		○	●				
Sacrées latérales			○	●	●	●	●
Fessière inf.						○	

les artères cervicales ascendantes thyroïdiennes inférieures et le tronc thyro-bicervico-scapulaire

Dans la région dorsale à partir des artères intercostales et pour les trois premiers segments par l'intercostale supérieure le tronc thyro-bicervico-scapulaire le tronc cervico-intercostal. Une à trois artères intercostales peuvent vasculariser la même vertèbre.

Dans les régions lombaires et sacrées (Tableau 2), à partir des artères lombaires sacrées moyennes ilio-lombaires et sacrées latérales

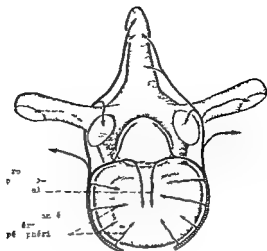


Fig 1 Vascularisation vertébrale

Il existe donc une vascularisation métamérique segmentaire des régions thoracique et lombaire. Au contraire, dans les régions cervicale et sacrée, ce caractère métamérique est moins net en raison de la disposition verticale des sources d'irrigation (vertébrale cervicale ascendante et profonde sacrée moyenne et latérale).

Aux confins de ces différents territoires, il existe un plus grand nombre de sources (C3 à D3 et L5 à S1).

*Les corps vertébraux.* La vascularisation a été particulièrement bien étudiée sur le plan anatomique par MARKHASHOV et nous reprendrons ici les données essentielles de son travail. La vertèbre est vascularisée par deux groupes d'artères (Fig 1).

1. Un groupe antéro-latéral périphérique. À la surface du corps vertébral, certaines artères pénètrent directement dans la vertèbre, d'autres forment des anastomoses soit avec le côté opposé, soit avec la vertèbre sus- et sous-jacente. Il existe ainsi à la surface antéro-latérale des corps vertébraux un réseau anastomotique qui se poursuit sur toute la longueur de la colonne vertébrale.

(2) Un groupe postérieur central. Il pénètre par le trou de conjugaison et alimente les parois et le contenu du canal rachidien (moelle et dure mère). Les artères se dirigent vers la face postérieure du corps vertébral et s'anastomosent avec les artères sus- et sous-jacentes et avec celles du côté opposé. Cette anastomose rétro-somatique est particulièrement bien vue à l'angiographie (Fig 2) et présente sur les clichés de face un aspect en M ou en H caractéristique.

Il existe également des branches pour les apophyses transverses et épineuses de



Fig 2 Anastomose arterielle retro-somatique Face soustraction L'aspect caracteristique et les anastomoses verticales avec les vertebres sus et sous jacentes (—>)

chaque cote (Fig 2) Les groupes antero-lateral et posterieur ont anastomoses et suivant la predominance de l'un ou de l'autre, on peut rencontrer d'apres MARASHOV differents aspects que nous avons schematise sur Fig 3 Cette double vascularisation s'explique par l'embryologie La partie centrale ou noyau d'ossification et la partie peripherique ou cartilagineuse ont des sources differentes d'irrigation sanguine et ne forment pas d'anastomoses Avec l'age, des anastomoses se developpent tant dans le centre des corps vertebraux que dans leur zone peripherique

Le nombre d'arteres intra somatiques penetrant dans les corps vertebraux varie chez l'adulte dans des limites importantes — 3 a 26 — selon le niveau de la colonne vertebrale en fonction de la taille du corps vertebraux Le nombre d'arteres intra somatiques suivant le niveau topographique est (d'apres MARASHOV) 6 a 15 arteres dans la region cervicale 12 a 21 arteres dans la region dorsale 17 a 26 arteres dans la regions dorsale basse et lombaire, et 3 a 13 arteres dans la region sacree Il est plus important dans les regions dorsale et lombaire ce qui explique vraisemblablement leur opacification plus frequente a l'angiographie



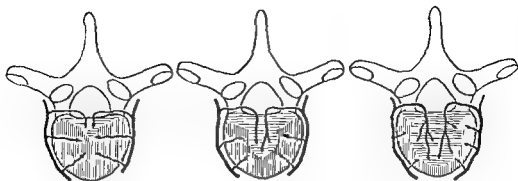


Fig 3 Variations dans la distribution et les anastomoses entre le groupe antéro latéral périphérique (||) et le groupe postérieur central (≡) (D'après MARILASOV)

### Radiologie

Nous avons étudié angiographiquement onze cas d'angiomes vertébraux (neuf dorsaux et deux lombaires), deux à forme douloureuse pure (dorsalgies douloureuses radiculaires) et neuf à forme paraplégique avec troubles sensitifs sphinctériens et blocage manométrique à la ponction lombaire.

L'aspect radiologique standard est bien connu et nous ne ferons qu'en rappeler l'aspect classique : soit micro lacunes bien délimitées donnant un aspect en nid d'abeilles soit fines stries verticales réalisant un aspect peigne.

Il faut souligner que les tomographies de face et de profil sont importantes — mais parfois insuffisantes — pour apprécier le degré d'envahissement de l'arc postérieur (pedicules et lames), du trou de conjugaison et du canal rachidien.

**Angiographie.** La technique utilisée est celle de l'angiographie médullaire (DJINDJIAN) sur les modalités de laquelle nous ne reviendrons pas. Pour chaque angiome nous avons opacifié sélectivement les deux artères nourricières droite et gauche (intercostales ou lombaires), ainsi que les artères sus- et sous-jacentes. La scintigraphie est étendue sur 30 secondes avec incidences de face et de profil, les clichés sont ensuite étudiés en soustraction.

Fig 4 Angiome vertébral dorsale (D5). Angiographie sélective des artères intercostales (→) droite (en haut) et gauche (en bas). Face soustraction. Aspects angiographiques à 1, 2 et 4 secondes après le début de l'injection. L'hypertrophie des artères du corps vertébral (↔↔)

Fig 5 Même cas. Profil soustraction. Clichés à 1, 2 et 4 secondes après le début de l'injection. Sur le cliché tardif on voit l'hématome épidural responsable de la compression médullaire sous la forme d'une opacité en arrière du corps vertébral (↔↔)

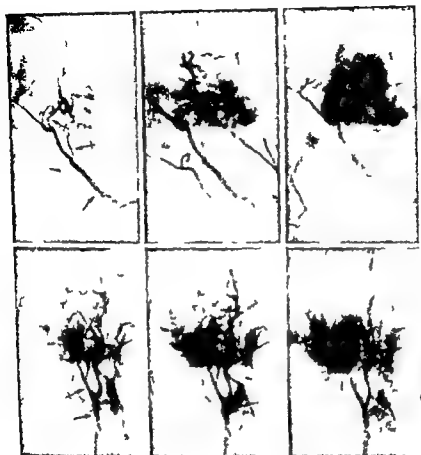


Fig 4 (Pour legende voir la page opposée )



Fig 5 (Pour legende voir la page opposée )



Fig 6 Angiome vertébral dorsal (D9) Face sous traction Aspect d'hémivascularisation L'angiographie de l'artère intercostale droite opacifie l'hémivertèbre droite

L'aspect angiographique est très particulier. Sur les clichés de face (Fig 4), il existe une opacification très précoce du corps vertébral dès la première seconde par une ou plusieurs artères antéro-latérales et postérieures. Ces vaisseaux nourriciers sont hypertrophiés, bien visibles et se jettent dans de nombreux lacis sanguins. L'opacification est dense, irrégulière, en plaques, avec volumineux néo-vaisseaux donnant à la vertèbre un aspect caverneux.

Le drainage veineux est précoce, mais la vertèbre reste opacifiée pendant longtemps sur la sériographie (jusqu'à 11 secondes). Le drainage se fait vers les veines intercostales et les veines de l'espace épidural.

Sur les clichés de profil (Fig 5), on retrouve le même aspect, mais la soustraction permet une étude très précise de l'extension de l'angiome à l'arc postérieur et surtout de l'association à un angiome épidural responsable de la compression, en relevant une opacité en arrière du corps vertébral et débordant l'image des pédicules.

Ces aspects angiographiques sont caractéristiques et ont été retrouvés d'une façon quasi-constante. Parfois cependant l'hypervascularisation pathologique paraît se localiser préférentiellement à l'hémivertèbre du côté injecté (deux fois sur onze cas) et donne l'impression de deux hémivascularisations relativement indépendantes (Fig 6).

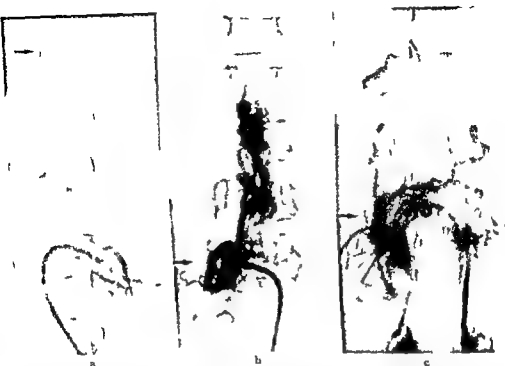


Fig 7 Angiographie a) Vertèbre dorsale (D12) d'apparence normale non angiomatueuse (—) b) Angiographie sélective de la 12ème artère intercostale droite. Angiome médullaire (—) apparaît discrètement hypervascularisé. On lui est difficile d'affirmer : cet aspect est-il anormal pathologique ou non c) Angiographie sélective de contrôle au même niveau et même totalité du corps vertébral de D12 (—) en donnant un aspect caractéristique d'angiome médullaire. Il est très vraisemblable que l'excès de l'angiome médullaire a permis une modification de la vertèbre pathologique en supprimant le vol sanguin à son niveau.

Nous n'avons jamais rencontré dans notre série d'association avec un angiome médullaire.

**Problèmes diagnostiques.** L'opacification par angiographie sélective des vertèbres nous amène à discuter un certain nombre de diagnostics différentiels.

(1) La vertèbre normale. Son opacification est homogène transitoire, limitée à l'hémi-corps vertébral correspondant au côté injecté mais sans dilatation ni hypertrophie des vaisseaux nourriciers sans aspect caverneux.

(2) Deux points particuliers ont à souligner. Certains angiomes intravertébraux sur les clichés standard ont injectés à l'angiographie (Fig 7). Ces angiomes correspondent certainement à la fréquence des angiomes vertébraux retrouvés par Schmitt et JUNGHAUS à l'autopsie. Dans une observation de notre série, il existait un angiome médullaire dans le même métamère (Fig 7).

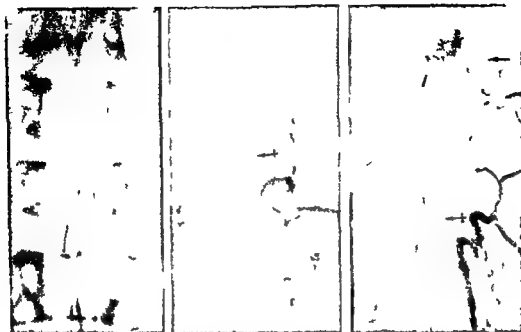


Fig 8 a

Fig 8 b

Fig 9

Fig 8 a) Vertèbre lombaire (L1) d'aspect angiomateux b) Angiographie soustraction Absence d'opacification du corps vertébral L'artère du renflement lombaire naît de la première artère lombaire gauche (→→)

Fig 9 Angiome vertébral dorsal (D9) Soustraction Opacification de l'angiome vertébral (→) L'artère du renflement lombaire (→→) naît de la 1<sup>ère</sup> artère intercostale gauche

Inversement il existe des vertèbres d'aspect angiomateux : grillage ou en nid d'abeilles sur les radiographies standard qui à l'angiographie ne s'injectent pas (Fig 8). Ceci permet d'éliminer un angiome vertébral vrai et pose peut-être le problème des autres affections osseuses notamment dysplasiques.

### Conclusion

Ainsi l'angiographie sélective permet une meilleure approche du diagnostic étiologique des vertèbres pathologiques, et un repérage précis des grands axes vasculaires fonctionnels. Son incidence thérapeutique est double.

(1) Elle permet de repérer en cas d'angiome osseux dorso-lombaire notamment (Fig 9) l'artère du renflement lombaire ou l'artère radiculo-médullaire dorsale haute qu'il faudra préserver, non seulement lors de la laminectomie décompressive mais aussi lors de la radiothérapie évitant ou diminuant ainsi le risque de radionécrose chimique.

(2) Elle permet de repérer les pédicules nourriciers de la malformation osseuse (artères intercostales ou lombaires) qui peuvent être ligaturées par voie antérieure par le chirurgien vasculaire lorsqu'on sait qu'il n'existe pas à ce niveau d'afférence médullaire importante cette ligature première précédant la laminectomie et visant à la rendre moins hémorragique Deux de nos malades ont subi double intervention avec succès (LAZORTHES et coll.)

## RÉSUMÉ

Les auteurs rapportent 11 cas d'angiomes vertébraux étudiés par opacification sélective des artères intercostales ou lombaires L'aspect angiographique est très caractéristique avec hypertrophie des vaisseaux nourriciers et aspect caverneux Les profils sont indispensables pour apprécier le retentissement médullaire Ces opacifications pathologiques sont très différentes de l'opacification transitoire des vertèbres normales mais les auteurs insistent sur le fait que certaines vertèbres d'aspect pseudo-angiomateux sur les clichés standard ne sont pas opacifiées à l'angiographie alors qu'inversement certains angiomes invisibles sur les clichés standard sont injectés à l'angiographie L'autre intérêt de l'exploration angiographique est le repérage des grands axes fonctionnels vasculaires de la moelle lorsqu'on envisage un traitement (laminectomie radiothérapie)

## SUMMARY

Selective angiography of the intercostal and lumbar arteries revealed eleven cases of vertebral angioma The widened nutrient arteries and cavernous appearances were characteristic features Lateral views are essential for an appreciation of the involvement of the spinal cord The angiographic signs are quite different from the transitory filling in normal vertebral bodies The authors stress that certain vertebrae presenting pseudo-angiomatous appearances in the ordinary film may remain unfilled at angiography while conversely angiography may be the only indication of an angioma A further advantage of angiography is that conditions of the spine may be related to their specific arterial supply which is important when considering whether the correct line of treatment should be laminectomy or radiation therapy

## ZUSAMMENFASSUNG

Mit Hilfe der Angiographie war es möglich elf Fälle von Hämangiom der Wirbel zu erkennen Das Röntgenbild mit den erweiterten Gefässkanälen und den Hohlräumen in den Wirbelkörpern ist charakteristisch Seitenaufnahmen sind wichtig um die Beteiligung des Rückenmarkes zu beurteilen Das pathologische Angiogramm ist grundverschieden von der flüchtigen Füllung die man normalerweise antrifft Es wird betont dass manche Wirbel die einen pseudo-angiomatösen Eindruck auf der Normalaufnahme machen bei der Angiographie sich also normal aufweisen und umgekehrt bei der Angiographie Angiome gefunden werden die die gewöhnliche Aufnahme nicht vermuten liess Die Angiographie ermöglicht es bei Rückenmarkserkrankungen das erkrankte Gebiet auf ein festumgrenztes Blutversorgungsgebiet zu lokalisieren, was bei therapeutischen Entscheidungen sei es Laminektomie oder Tiefenbestrahlung von Nutzen ist

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## ARTÈRES DE LA DURE-MÈRE RACHIDIENNE CHEZ L'HOMME

par

C MANELFE G LAZORTHES et J ROULLEAU

La dure mere rachidienne ayant ete longtemps consideree comme une membrane inerte et fibro-élastique remplissant essentiellement une fonction mecanique sa vascularisation est restee dans l'oubli.

Les vaisseaux de la dure mere encephalique au contraire, ont fait l'objet de recherches approfondies des la deuxieme moitie du XIX<sup>eme</sup> siecle (BOEHM 1869 PASCHKEWICZ 1871 MICHEL 1872 KEY & RETZELS 1875, LANGER 1877).

Il fallut attendre 1930 pour que PFEIFER donne une representation a laquelle on a peu ajoute depuis, de l'angioarchitectonie de la dure mere cranienne. Les études d'IZMAILOVA (1953) et de VASEN (1959) constituent une derniere mise au point sur l'anatomie vasculaire microscopique de la dure mere cranienne. L'anatomie macroscopique et radiologique des vaisseaux de la dure mere encephalique a donne lieu a de nombreux travaux tant en France par GRISOLI (1966), SALAMON et coll (1967) qu'a l'etranger par GREITZ & LAUREN (1968) puis NEWTON (1968) sur les arteres meningees de la jonction cervico-occipitale.

Aussi la vascularisation de la dure mere rachidienne fait-elle figure de « parent pauvre », vis-a-vis de son homologue la dure mere cranienne et les traites d'anatomie actuels n'en donnent aucune description. C'est a la precision de l'origine, du mode de distribution et de terminaison ainsi qu'a la structure histologique des





Fig. 1. Dure-mère rachidienne ouverte sur sa face antérieure. Une artère spinale antérieure chemine sur la cinquième racine dorsale droite et perfore la dure-mère par un orifice sus-jacent à la racine. Nombreuses artères dure-mériennes longues injectées vues en transparence (→).

artères dure-mériennes (ou durales) chez l'homme que nous sommes attachés (MANELFE 1969).

*Matériel et méthodes.* Notre matériel d'étude porte sur une série de 20 sujets qui se répartissent de la façon suivante: 3 fœtus (1 de 6 mois, 2 de 7 mois) et 17 adultes (5 hommes, 12 femmes, l'âge variant de 51 à 88 ans). Nous avons volontairement éliminé de notre étude tout sujet décédé d'une affection cardiovasculaire majeure, d'une affection neurologique — notamment médullaire — ou vertébrale.

L'injection du produit de contraste (sulfate de baryum) est faite par voie générale non sélective dans l'artère fémorale chez l'adulte, dans l'aorte après ligature du canal artériel, des artères pulmonaires et des veines caves chez le fœtus.

La dure-mère rachidienne est prélevée après laminectomie large étendue du trou occipital au sacrum et les racines rachidiennes sont sectionnées le plus loin possible dans le trou de conjugaison. Après fixation dans une solution formolée et dissection, les prélèvements ont fait l'objet d'une quadruple étude: microdissection, radiographie et microradiographie, diaphanisation et histologie.

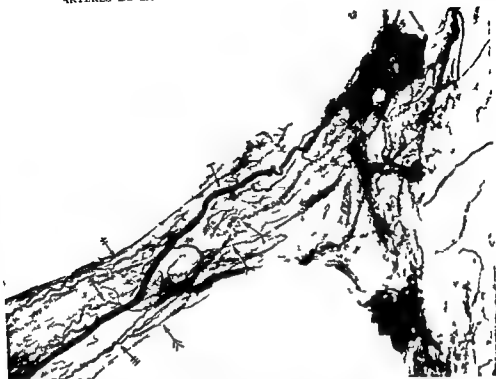


FIG. 2 Arteres dure meniennes courtes Branches directes et recurrentes Nombres arteres spirales (→) dont certaines mesurent pres de 10 mm de long L'artere radicaire (→) ne perforé ici la gaine durale qu'au niveau de son insertion sur le fourreau dural Artere perforant la dure mere (→) arteres courtes recurrentes (→) arteres courtes directes (→) anastomose (→) Grossissement 6 fois

### Resultats

La vascularisation arterielle de la dure mere rachidienne provient des arteres radiculaires dont les origines ont differentes suivant les territoires cervical dorsal et lombo-sacre)

Ces vaisseaux pairs et symetriques a disposition metamérique penetrent dans le canal rachidien au niveau des trous de conjugaison en accompagnant chaque nerf rachidien C'est a ce niveau que naissent les arteres dure meniennes et nous avons essaye d'en preciser l'origine le trajet et la systematisation

Les arteres dure meniennes ont de fins vaisseaux qui cheminent a la surface externe de la dure mere rachidienne et de ses gaines radiculaires auxquelles ils sont amarrés par de courts tractus fibreux

*Origine* Elles naissent des arteres radiculaires au cours de leur trajet a la surface de la gaine durale et toujours nous a-t-il semblé, avant la perforation de la dure mere par les arteres radiculaires. Nous n'avons pas trouve d'artere dure-

Fig 3 Arteres dure meniennes moyennes Branches de l'artere radiculaire l'une chemine pres du bord superieur de la gaine durale l'autre suit son bord inferieur Artere perforant la dure mere ( $\rightarrow$ ) artere moyenne ( $\leftrightarrow$ ) artere courte ( $\longrightarrow$ ) artere radiculaire ( $\dashrightarrow$ ) Grossissement 6 fois



menienne naissant le long du trajet sous-dural ou plus précisément entre la dure mere et l'espace sous-arachnoïdien radiculaire (Fig 1)

*Trajet et distribution* Ces arteres constituent un reseau complexe et riche. Les variations sont importantes et il est souvent difficile de dire s'il s'agit de branches collaterales ou d'un mode de terminaison particulier. En etudiant soigneusement nos pieces en microdissection et en microradiographie il nous a paru possible de scinder l'etude de ces arteres dure meniennes en trois types : les arteres dure meniennes courtes, moyennes et longues.

D'autre part, signalons des l'abord que deux formations ont particulierement retenu notre attention par leur caractere d'originalite : les arteres spirales ou en meandres et les pelotons vasculaires.

**1 Les arteres dure meniennes courtes** (Fig 2) Elles naissent soit de l'artere radiculaire, soit des arteres dure meniennes longues ou moyennes. Ce sont les plus nombreuses (cinq à dix en moyenne par racine), les plus fines et elles existent sur toutes les racines. Leur trajet, rectiligne ou tres sinueux, en spirale ou en meandres est soit direct soit recurrent vers le trou de conjugaison. Elles vascularisent essentiellement la gaine durale radiculaire entourant le nerf rachidien et ses racines.

**2 Les arteres dure meniennes moyennes** (Fig 3) Elles ont la meme origine que les precedentes mais s'en different par leur calibre legèrement superieur ( $120$  à  $150 \mu$ ) et par leur trajet plus long. Leur nombre varie mais le plus



Fig 4 Arteres dure meningeennes longues. Artere longue (○→) Division en branches ascendante (↑→) descendante (↓→) et transverse (→→) pour la face posterieure et en branches antero-superieure (↗→) et antero-inferieure (↘→) pour la face anterieure. Multiples anastomoses entre les differentes branches d'un meme cote et du cote oppose. Grossissement 25 fois.

souvent il en existe deux ou trois par racine. Elles suivent le bord superieur ou inferieur de la racine et se divisent frequemment en trois branches : une verucule a pendante ou de pendante deux transversales, dont l'une se dirige vers l'avant l'autre vers l'arriere mais sans jamais atteindre la ligne mediane. Elles vascularisent la gaine radiculaire et la dure mere rachidienne adjacente a la racine.

3 *Les arteres dure meningeennes longues* (Fig 4). Elles apparaissent comme les veritables arteres de la dure mere rachidienne etant les plus importantes par leur calibre (400  $\mu$  a l'origine) et par leur longueur. Elles naissent de l'artere radiculaire et sont moins nombreuses que les precedentes (une a deux par racine). On les rencontre pratiquement sur toutes les racines. Leur trajet suit la gaine radiculaire et elles se divisent en plusieurs branches terminales : ascendante descendante et transverse qui vascularisent la partie postero-laterale de la dure mere antero-superieure et antero-inferieure, qui vascularisent la partie antero-laterale de la dure mere. Toutes ces branches cheminent elles aussi sur la face externe du fourreau dural.

Leur mode d'origine et de trajet est variable et nous avons represente sur un schema les types de division les plus frequemment rencontres (Fig 5).

*Systématisation et voies anastomotiques*. La face posterieure de la dure mere rachidienne presente une riche vascularisation alors que la face anterieure est beaucoup plus pauvre. Les grands axes arteriels sont d'ailleurs differents sur les deux faces.

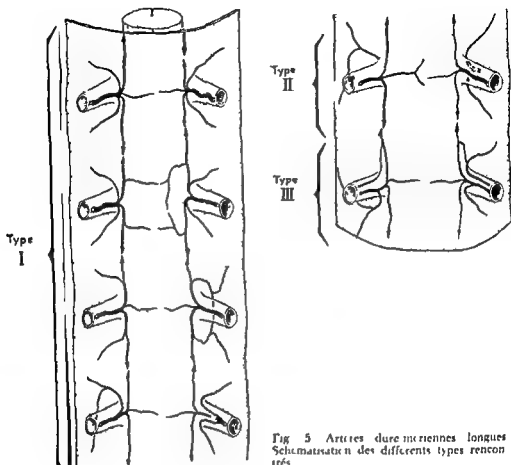


Fig. 5. Artères dure-mériennes longues. Schématisation des différents types rencontrés.

Sur la face antérieure il existe un courant artériel médian — ou très près de la ligne médiane — disposition qui rappelle celle de la voie artérielle spinale antérieure.

Sur la face postérieure — sauf pour les deux tiers supérieurs de la région cervicale (Fig. 6 a) — la vascularisation se concentre en deux courants artériels latéraux (Fig. 6 b et c) : cette disposition est comparable ici aussi aux deux voies spinales postéro-latérales.

Les artères dure-mériennes ont très richement anastomosées. À la surface externe de la dure-mère donnant une disposition en arcades (Figs 4, 5, 6 b et c) ou convergente (Fig. 6 a). Dans la profondeur de la dure-mère par des anastomosées en réseau de type plexiforme particulièrement développées comme au niveau de la dure-mère crânienne (PFEIFFER).

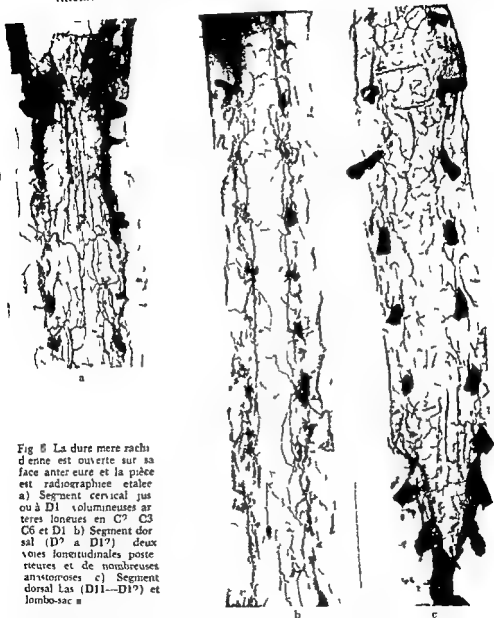


Fig 6 La dure mere rachidienne est ouverte sur sa face anterieure et la piece est radiographiée etalée  
 a) Segment cervical jus-  
 qu'à D1 volumineuses ar-  
 teres longues en C<sup>7</sup> C3  
 C6 et D1 b) Segment dor-  
 sal (D<sup>7</sup> a D1<sup>9</sup>) deux  
 voies longitudinales poste-  
 rieures et de nombreuses  
 anastomoses c) Segment  
 dorsal Las (D11—D1<sup>9</sup>) et  
 lombo-sac ■

Ces différentes anastomoses ont très nombreuses mais il est difficile de pre-  
 juger de leur réelle valeur fonctionnelle

Chez le fœtus (Fig 7) Bien que le nombre très réduit des sujets étudiés ne  
 permette d'avancer aucune conclusion définitive nos constatations ont identiques  
 dans le 3 cas



Fig 7

Fig 8

Fig 7 Dure-mère rachidienne (le pus de l'œuf et d'un segment dorsal D1 à D8). L'importante vascularisation de la face postérieure par rapport à la face antérieure.

Fig 8 Arteries spirales en méandres. Quatrième segment dorsal. C'est ainsi ment (ici).

La vascularisation est comparable à celle de l'adulte. On note ici aussi (1) une prédominance de la vascularisation sur la face postérieure et (2) un réseau anastomotique déjà développé avec individualisation des voies longitudinales postérieures.

Ainsi la vascularisation de la dure-mère rachidienne obéit-elle à une distribution segmentaire métamérique et présente des caractères nettement différents de la vascularisation médullaire. Il ne paraît pas exister de territoire vasculaire pauvre, dans la région dorsale moyenne (D4 à D8) notamment. Elle se rapproche au contraire beaucoup plus de la vascularisation métamérique de la colonne vertébrale.

### Les artères spirales ou «en méandres» et les pelotons vasculaires

Nous avons été frappés par l'existence de deux formations particulières sur la dure-mère rachidienne : les artères spirales et les pelotons vasculaires. Les ayant déjà décrites dans un précédent travail (LAZORTHES & MANELFE 1970), nous n'en donnerons que les principaux caractères.

**Les artères spirales ou «en méandres»** (Fig 8). Elles sont branchées sur des collatérales ou terminales (1), au niveau des artères longues le plus souvent. Leur diamètre variable (1 à 10 mm) leur trajet est le plus souvent



Fig 9



Fig 10

**Fig 9** Peloton vasculaire. Deuxième segment dural dorsal. Il est formé de très nombreuses circonvolutions situées dans les trois plans de l'espace. On voit une artère afférente et peut-être une seconde parallèle plus grêle (ou branche collatérale). Du côté opposé à l'artère afférente on aperçoit une veine quittant le peloton (→). Grossissement 13 fois.

**Fig 10** Angiome médullaire dorsal bas au du cône terminal alimenté par une afférence artérielle unique en D12 droite. On voit en regard de D12—D11 et D10 du côté gauche une artère dure mérienne longue dilatée dont la partie terminale présente un trajet spirale caractéristique (→). (Observation R. Djindjian.)

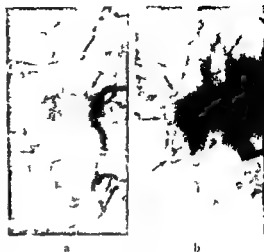
vertical ou oblique. On les retrouve sur les gaines radiculaires, les faces — surtout postérieure et latérale — de la dure mère toujours sur sa face externe dans un dedoublement conjonctivo-fibreux ou dans son épaisseur mais jamais sur la face interne.

Leur structure histologique ne présente aucune particularité par rapport aux artères durales proprement dites.

**Les pelotons vasculaires** (Fig 9). Ils sont formés par un ou plusieurs artères afférentes aboutissant à un paquet vasculaire qui présente un aspect vermiforme. Ils sont terminés par une véritable pelote vasculaire formée par une veine. La taille de ces pelotons est de 1 à 2 mm.



Fig 11 Méningiome dorsal a) L'angiographie sélective de la 8<sup>ème</sup> artère intercostale gauche opacifie une masse ovale par l'intermédiaire d'une artère dure mérienne hypertrophiée (→) b) Vaisseau bordant circonscrivant le bord interne de la tumeur (→) (Observation R. Djindjian)



On les trouve principalement sur la face postérieure de la dure mère rachidienne — surtout dans la région dorsale — exceptionnellement sur la face antérieure. Ils sont toujours situés sur la face externe de la dure mère faisant saillie dans l'espace epidural.

*Discussion* Ces deux types de formations étudiées au niveau de la couche perostée de la dure mère crânienne par KEY & RETZIUS en 1875 et récemment par HAMMERSEN & STAUBESAND en 1963 n'ont jamais été décrits au niveau de la dure mère rachidienne.

Les vaisseaux spirales existent, on le sait au niveau de très nombreuses séreuses et organes ovaire, uterus, cordon spermatique, rein et appareil juxta glomérulaire.

On peut se demander quel est le rôle physio-pathologique de ce phénomène de « spiralisation » ou d'aspect « en méandres » des vaisseaux. Il ne paraît pas lié à un simple vieillissement artériel car LANGER (1877) a constaté un aspect identique sur la dure mère crânienne de l'enfant.

On se demande aussi s'il s'agit de formations permanentes ou temporaires. On sait qu'ils peuvent apparaître au voisinage de processus inflammatoires (DE LANGEN 1964) ou lors de l'oblitération d'un vaisseau (DOMINI 1957, TAGARIELLO & DOMINI 1958), DOMINGUEZ (1955) leur attribue une fonction de réservoir, TAGARIELLO & DOMINI y voient plutôt un dispositif régulateur de courant. HAMMERSEN & STAUBESAND considèrent qu'en aucune façon n'incombent des fonctions hémodynamiques spéciales aux artères spirales car d'après eux elles ne seraient que l'expression de besoins vasculaires antérieurs.

Néanmoins ces différentes conceptions n'expliquent pas pourquoi ces artères ne se développent que par endroits et sous des aspects aussi remarquablement divers.

Quant aux pelotons vasculaires, HAMMERSEN & STAUBESAND en font, à l'étage crânien, des dispositifs régulateurs du flux sanguin.

Ici les rapports de contiguïté des artères et des veines font élever l'hypothèse d'anastomoses artério-veineuses à ce niveau mentionnées par ALPRTAHOV. Nous avons constaté, sur certaines préparations de pelotons vasculaires, la présence dans les veines de particules de sulfate de baryum. On sait qu'en principe ce produit ne dépasse pas la barrière précapillaire et ne doit pas être retrouvé dans les veines. À moins d'un artefact technique toujours possible, on peut voir ici la preuve de l'existence, au niveau des pelotons vasculaires, d'anastomoses artério-veineuses. Il reste à élucider si ces formations jouent un rôle dans la circulation ou la résorption du L.C.R.

### Etude angiographique

Sur le plan pratique, on peut voir au cours de l'angiographie médullaire les vaisseaux de la dure mère rachidienne. Leur faible calibre (très peu dépassent 0,5 mm de diamètre) interdit jusqu'à présent leur visualisation *in vivo*.

Cependant, dans certaines conditions pathologiques, ces vaisseaux peuvent se hypertrophier de façon considérable. L'angiographie médullaire selon la méthode de DJINDJIAN permet de les mettre en évidence (1) dans les tumeurs vasculaires, les rares hémangiomes caverneux de la dure mère, certains angiomes médullaires (Fig. 10) et (2) dans les méningiomes extra-duraux (Fig. 11). Il ne faut pas les confondre avec les artères destinées au corps vertébral, nees, elles aussi des artères radiculaires. L'aspect spirale des vaisseaux dure-mériens paraît assez caractéristique.

### Conclusion

L'anatomie des artères de la dure mère rachidienne était jusqu'à présent inconnue et nous avons essayé d'en décrire et d'en préciser les principales caractéristiques. Un certain nombre de constatations restent encore à éclaircir, notamment en ce qui concerne les vaisseaux spirales ou en méandres et les pelotons vasculaires. Ils jouent peut-être un rôle dans la dynamique du L.C.R. à l'étage rachidien.

## RÉSUMÉ

Les auteurs présentent une étude radio anatomique originale de la vascularisation artérielle de la dure mère rachidienne chez l'homme. La distribution vasculaire est segmentaire métamérique à partir des artères radiculaires et la face postérieure du fourreau dural est plus richement vascularisée que la face antérieure. Le fait marquant de cette étude est l'existence de formations vasculaires très particulières (vaisseaux spirales ou « en méandre » pelotons vasculaires) dont la signification physiologique reste encore inconnue. Ces artères dures mérielles peuvent dans certaines conditions pathologiques (tumeurs vasculaires méningiomes) s'hypertrophier et être ainsi visualisées par l'angiographie médullaire.

## SUMMARY

A roentgen investigation of the anatomy of the arterial supply system of the human spinal dura mater is presented. This supply is derived from the radicular arteries and is of a segmental nature. The dural sheath is more vascular posteriorly than anteriorly. A new and remarkable feature emerged in the form of peculiar vessels of meandering course or in the form of whorls the function of these vessels remains obscure. The dural vessels may be hypertrophied in conditions such as vascular tumours or meningiomas; this may be detected by angiography of the spinal cord.

## ZUSAMMENFASSUNG

Eine Untersuchung der Röntgenanatomie der Gefäße des Rückenmarkes wurde unternommen. Es zeigte sich, dass die Gefäße segmentartig angeordnet sind und von den radikulären Arterien entspringen. Die Hinterseite der Durahülle ist gefäßreicher als die Vorderseite. Neu entdeckt wurden besondere Gefäßbildungen, die einen spiralartigen oder meanderförmigen Verlauf hatten oder Gefäßknäuel bildeten. Die Funktion dieser Gefäße ist unklar. Unter bestimmten pathologischen Bedingungen z. B. bei gefäßreichen Tumoren oder Meningeomen können die Duralgefäße eine Hypertrophie im Angiogramm aufzeigen.

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## LUMBALFUNKTIONSMYELOGRAPHIE

VON

H MOSL, H E DIEMATH, W D ORTNER, H KUBIN, W A F KOLLAR und  
J STROHECKER

Der zusätzliche Begriff Funktion beinhaltet daß bei einer lumbalen Myelographie — eigentlich richtiger ausgedrückt — bei einer lumbosakralen Radikulographie, vornehmlich für die Diagnostik von Bandscheibenhernien auch die Statik und Dynamik des Wirbelsäulenabschnittes und des dargestellten Inhaltes des Spinalkanales mit aufgezeichnet werden sollen. Dieses Ziel ist zu erreichen wenn die Untersuchung im Stehen durchgeführt werden kann. Damitcheiden in der Wahl der Kontrastmittel solche welche eine Lumbalanästhesie verlangen, aus Gas ist schon aus physikalischen Gründen nicht verwendbar. Von Präparaten vom Typ der Jodester wurde bei einer Untersuchung im Stehen eine relativ große Menge zur zusammenhängenden Darstellung der unteren und mittleren lumbalen Etagen benötigt werden, außerdem ist ihre Kontrastdichtigkeit für eine Radikulographie zu groß.

Für die Diagnostik lumbaler Diskushernien hat sich uns Conray 60 bisher am besten bewährt. Das Kontrastmittel kann ohne Lumbalanästhesie in den Subarachnoidalraum instilliert werden. Der Patient bleibt während der Unter-

sung, die rasch und ohne Zeitdruck ausgeführt werden kann beweglich. Nach 4 bis 6 Stunden ist das Kontrastmittel meist vollständig reorbiert. Über Nebenwirkungen und Komplikationen sind bereits mehrere Publikationen erschienen. Unsere Erfahrungen stimmen mit denen des Schrifttums überein.

**Untersuchungstechnik** Lumbalpunktion möglichst unter L2/L3 im Sitzen. Injektion eines Gemisches von 4 ml Liquor und 6 ml Conray 60 subarachnoidal unter Durchleuchtungskontrolle, um ein Ansteigen der Kontrastmittelsäule bis L1 und höher möglichst zu vermeiden, was insbesondere bei Vorliegen eines Stops der Fall sein kann. Nach Entfernung der Punktionsnadel werden im Stehen unter Fernschrägdurchleuchtung Zielaufnahmen in folgenden Positionen angefertigt: (1) eine orthograde a.p. Aufnahme, (2) eine a.p. Aufnahme bei Seitenneigung des Patienten nach rechts, (3) eine a.p. Aufnahme bei Seitenneigung des Patienten nach links, (4) eine oder zwei Schrägprojektionen in Hyperlordose zur Darstellung der Wurzeln der kranken Seite im Profil, (5) eine Schrägprojektion in Hyperlordose zur Darstellung der Wurzeln der Gegenseite und (6) je eine seitliche Aufnahme in Hyperlordose und in Ventralflexion der Wirbelsäule.

Die Durchleuchtung mittels der Bildverstärker-Fernkette erfolgt nur kurz orientierend zur Einstellung der eng einzubildenden Zielaufnahmen, um die Strahlenbelastung so gering wie möglich zu halten.

Schon während des Untersuchungsvorganges werden die Filme in eine 90 sec. Entwicklungsmaschine eingebracht, sodaß nach Beendigung der Untersuchung schon die ersten Bilder begutachtet werden können und Korrekturen z. B. wegen einer Bewegungsunschärfe, rasch möglich sind. Anschliessend muß der Patient auf einem Stuhl oder im Bett 6 Stunden sitzen, um den Kontakt des mit der Liquorzirkulation aufsteigenden Kontrastmittels mit dem Rückenmark möglichst klein zu halten.

Die Bildserie (Abb. 1) zeigt die Ergebnisse einer mit dieser Technik durchgeführten lumbosakralen Radikulographie. (Die Aufnahme in Schrägprojektion zur Darstellung der Wurzeln der gesunden Seite wurde absichtlich weggelassen.)

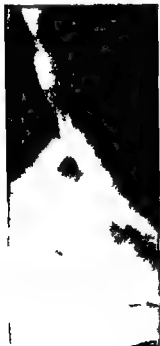
Besondere Bedeutung erlangt die Funktionsuntersuchung in der Differentialdiagnose zwischen einer medianen Hernie und einer dorsalen Diskusprotrusion. Die ventrale Eindellung des Kontrastbandes, welche durch eine Protrusion verursacht wurde, schwindet bei Streckung der Lendenlordose; bei einer Hernie bleibt sie bestehen, beziehungsweise wird sie nur etwas flacher.

Auch ein pendelnder Prolaps dürfte sich im Stehen durch Provokation in Hyperlordose eventuell unter Anspannung der Bauchpresse oft darstellen lassen (Abb. 2).

Ferner kann bei der von uns geübten Aufnahmetechnik die Verformbarkeit



a



b

Abb. 1 (Legende auf der geg. gegenüberstehenden Seite)

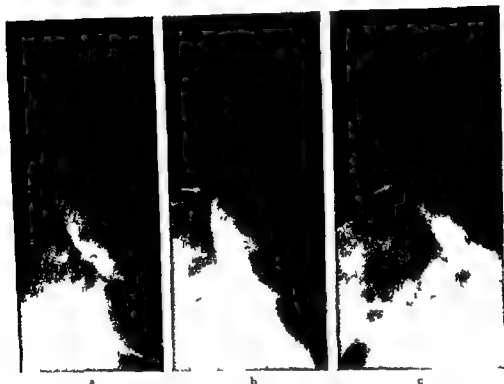


Abb 2 Dorsale Diskusprotrusion bei L3/L4 In Mittelstellung (a) keinerlei Eindellung am Kontrastband in Hyperlordose (b) deutliche ventrale Impression bei L3/L4 In maximaler Ventralflexion (c) ist diese Eindellung völlig verschwunden

der Zwischenwirbelräume und deren Anpassung an die Beweglichkeit beurteilt und der aktuelle Zustand einer Skoliose hinsichtlich einer noch vorhandenen Mobilität geprüft werden

Die für eine Spondylolisthesis typische Beweglichkeit und ihre Rückwirkung auf den Inhalt des Spinalkanals kann bei einer Funktionsradikulographie im Stehen besser als bei Schraglage des Patienten eingeschätzt werden (Abb 3)

Wir haben bei 100 Conray Myelogrammen zusätzlich eine Aufnahmeserie in Schraglage des Patienten (30° über der Horizontalen) mit gleichen Projektionen

Abb 1 Medio-laterale Bandscheibenhernie in Höhe L5/S1 mit einer Kompression der rechten Wurzel von S1. a) Diese bleibt bei Rechtsbeugung erhalten bei Linksbeugung ist dieser Befund etwas weniger deutlich b) In der Schragprojektion in Hyperlordose ist neben der Verkürzung der Wurzeltasche von S1 und einer Verbiegung der Wurzel auch eine leichte Impression der Wurzel von S<sub>2</sub> sichtbar Sowohl in Hyperlordose als auch in Ventralflexion bleibt die ventrale Impression in Höhe des Bandscheibenraumes L5/S1 erhalten eine geringe solche ist auch in Höhe des Bandscheibenraumes L4/L5 vorhanden





Abb 3 Spondylolisthesis L4/L5 In Hyperlordose Verstärkung sagittalen Wirbelgliedens von L4 und der ventralen Impression Kontrastbandes im Vergleich zur Ventralflexion

wie bei der Untersuchung im Stehen angefertigt. Ein Vergleich zeigte, daß bei 73 röntgenologisch positiven Fällen bei 22 Patienten (30,1 %) der Befund im Funktionsmyelogramm deutlicher dargestellt werden kann. Daraus geht aber auch hervor, daß in keinem Fall bei alleiniger Untersuchung in Schräglage des Patienten 30° zur Horizontalen bei gleichen Projektionen und Provokation in Hyperlordose die Diagnose einer Diskushernie nicht hätte gestellt werden können. Bei einer Untersuchung im Stehen tritt nur wie Abb. 4 zeigt, der Befund deutlicher hervor.

Der Wert der aufgezeigten Untersuchungstechnik, besonders für die Diagnostik lumbaler Diskushernien, liegt darin, daß (1) auch kleine Hernien deutlicher erfasst werden können, (2) die Beweglichkeit der Wirbelsäule und die Verformung der Bandscheibenräume mitstudiert werden können, (3) die Untersuchung rasch und ohne Lumbalanästhesie durchzuführen ist und (4) bei unklarem Befund sofort weitere Aufnahmeserien angefertigt werden können.

Ein Nachteil sind lediglich die durch das Kontrastmittel hervorgerufenen Nebenwirkungen und Komplikationen, die leider so lange in Kauf genommen werden müssen, bis das ideale Kontrastmittel gefunden ist.



Abb 4 a) A p Projektion in Schraglage des Patienten (30° über der Horizontalen) Leichte Abplattung der Wurzel tasche L5 in Höhe des Bandscheibenraumes L4/L5 b) Gleiche Position des Patienten nur nach rechts aufgedreht Zusätzlich ist noch eine flache Impression der rechten Wurzel tasche von S1 sichtbar c) Bei starkerer Aufdrehung ist der Befund nicht deutlicher d) Schragprojektion im Stehen bei Hyperlordose Fast subtotaler Stop mit Kompression mehrerer Wurzeln in Höhe des Bandscheibenraumes L4/L5

## ZUSAMMENFASSUNG

Die Autoren berichten über die Technik und den Wert der lumbalen Funktionsmyelographie zur Diagnostik von Diskushernien mit Conray 60 Eine vergleichende Auswertung von in konventioneller Technik durchgeführten Myelographien und der von ihnen geübten Untersuchungsmethode zeigt die bessere Darstellung auch kleinerer Hernien

## SUMMARY

The authors describe their technique for functional lumbar myelography with Conray 60 in the diagnosis of disc prolapse A comparison of the new technique with the old procedure indicates that the new method yields better results especially in the smaller disc herniations

## RÉSUMÉ

Les auteurs étudient la technique et l'intérêt de la sacroradiculographie fonctionnelle au Conray 60 pour le diagnostic des hernies discales La comparaison des radiculographies faites par la technique habituelle et de sacroradiculographies faites au leur méthode d'examen montre que celle-ci met en évidence les petites hernies

## WATER-SOLUBLE CONTRAST MEDIUM MYELOGRAPHY IN THE EXPERIMENTAL ANIMAL

A preliminary report

by

J PRESTHOLM

A comparative investigation of the use of the methyl glucamine salt of iothalamate (Conray Meglumin 282 Conray 60 %, Contrix 28 %) and the methyl glucamine salt of dimersed iothalamate (MP 2032 Mallinckrodt) in myelography in rabbits has been performed

KODAMA et coll (1963) observed a higher LD<sub>50</sub> with iothalamate meglumine than with other water soluble contrast media following intracisternal injection in rabbits. FISHER (1965) carried out experiments with iothalamate meglumine 60 % in doses of 0.25 and 0.5 ml per kg body weight in lumbar myelography in cats under Nembutal anaesthesia. All the cats became hyperpneic and several lost urine and feces during the injection. They were killed from one week to three months after the myelography and histologic examination revealed no changes in the spinal cord or meninges.

HEIMBURGER et coll (1966) employed dogs under Nembutal anaesthesia. They injected iothalamate meglumine 60 % intraventricularly into 13 dogs and intracisternally into 87 dogs in doses varying from 1 to 15 ml (63 to 700 mg

dry matter per kg body weight) With the larger doses the dogs died with evidence of raised intracranial pressure. Convulsions developed with the lower doses, the LD<sub>50</sub> was calculated to be 355 mg/kg. The authors carried out histologic examinations of the brains of those dogs that survived the acute stage and were killed after one week or one month. No microscopic abnormalities were observed.

HILAL (1966) was the first to employ dimerised iothalamate. He stated that this contrast medium produced fewer adverse reactions than others in angiography.

GONSETTE & ANDRÉ BALISALY (1970) have recently described experiments in which they applied both iothalamate meglumine and dimerised iothalamate meglumine intrathecally to one hemisphere of the brain of guinea pigs in a dose of 0.05 ml. The convulsive properties of the two contrast media were compared, and the brains were examined by both optical and electron microscopy. It was concluded that only dimerised iothalamate meglumine presents des garanties d'innocuité suffisante pour autoriser des essais cliniques chez l'homme. The authors stated that their results would be reported in detail in a subsequent article.

*Material and Methods* The experiments were carried out in rabbits of 3 kg average weight. Myelography was performed under intravenous Nembutal anaesthesia (initial dose of 30 to 40 mg Nembutal per kg body weight). The contrast medium in a volume of 0.4 ml was injected into the subarachnoid space by suboccipital puncture before which 0.3 to 0.5 ml cerebrospinal fluid was aspirated. Films were obtained of the skull and entire vertebral column after the injection to control the correct positioning of the contrast medium. The CSF was examined for protein and cell content: the former by Lowry's micro-method (Lowry et coll. 1951). The rabbits were killed under Nembutal anaesthesia 24 hours, 7 days, 3 weeks or 6 weeks after the myelography by bleeding after cutting both carotid arteries. Suboccipital puncture was then immediately performed with aspiration of from 0.3 to 0.6 ml CSF. At autopsy the vertebral column was dissected out and the laminae removed, the spinal cord remained in situ on the vertebral bodies and the entire preparation together with the brain, were fixed in formalin 10% for at least 3 weeks. Samples were removed from a total of 12 segments of the spinal cord evenly distributed from C1 to S3 for the histologic examinations. The following four histologic staining methods were used: haematoxylin-eosin, van Gieson, Hansen's connective tissue stain, Luxol Fast Blue myelin stain by the Kluver-Barrera method and Lissac's galloxyanine-chromalum stain for chromatin.

The actual part of the material comprised 90 rabbits, 28 of which received



Fig. 1. Myelography in a rabbit after suboccipital injection of 0.6 ml Conray Meglumin 60 %.

0.4 ml Conray Meglumin 30 %, 34 had 0.4 ml MP 2032 35 %, while 28 rabbits served as controls and received no contrast medium. Suboccipital puncture was carried out in 8 of the control animals before they were killed, while the remaining 20 rabbits underwent no form of operation.

### Results

The myelography was incomplete with 0.4 ml Conray Meglumin 30 % and 0.4 ml MP 2032 35 %. Characteristically there was moderately good contrast outline from C1 to C7 and poor delineation from Th10 to L6. Contrast medium never appeared intracranially. A clear outline of the entire spinal subarachnoid space was obtained in another series with 0.6 ml Conray Meglumin 60 % or 0.6 ml MP 2032 70 % (Fig. 1).

All the rabbits developed convulsions after myelography with the exception of 2 that received 0.4 ml MP 2032 35 %. These started with reflex spasm of the forelegs in response to touch and gradually extended to bouts of spasm of all four limbs: episthotonus and extensor rigidity. It was necessary to maintain the anaesthesia so long as there was a tendency to convulsions, as otherwise the muscle rigidity interfered with respiration, and the spasms led to fractures of the spine. Five rabbits died during myelography after the injection of 0.4 ml Conray Meglumin 30 % or MP 2032 35 %: 2 due to an overdose of Nembutal and 3 in convulsions caused by underdoses of the anaesthetic. Convulsion fractures of the spine occurred in 2 rabbits that had received Conray Meglumin and in one rabbit that had had MP 2032. The following values were used to give a quantitative measure of the convulsion provoking properties of the two contrast

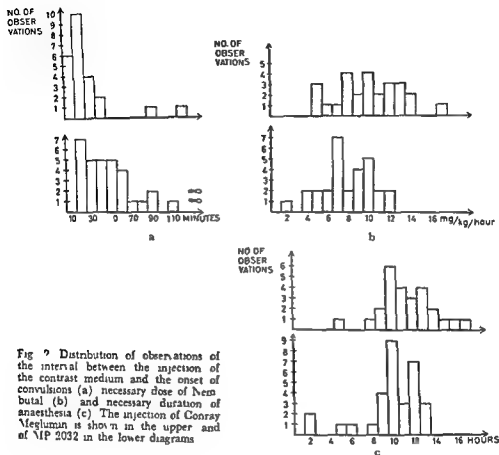


Fig 2 Distribution of observations of the interval between the injection of the contrast medium and the onset of convulsions (a) necessary dose of Nembutal (b) and necessary duration of anaesthesia (c) The injection of Conray Meglumine is shown in the upper and of MP 2032 in the lower diagrams

media (1) the length of time between the injection of the contrast medium and the onset of the convulsions, (2) the dose of anaesthetic and (3) the necessary duration of anaesthesia. The distribution of these values in 28 rabbits that had received 0.4 ml Conray Meglumine 30% and 34 rabbits that had had 0.4 ml MP 2032 30% is evident from Fig 2. A few rabbits have been omitted from one or more of the diagrams either because they died during the experiment or because of uncertainty in the registration of the data. Fig 2a indicates that the observations follow an asymmetric distribution and it is therefore impossible to use normal distribution analyses in the statistical evaluation of the values. Wilcoxon's rank sum test is employed instead. The numbers of observations in the two groups were 24 and 33. The rank sum of the first group is 173. As the 2 per cent limits for the distribution are (537-855) the hypothesis that the same distribution exists in both groups is rejected at the 2 per cent level. The

Table 4

*Histologic changes after myelography in rabbits related to the contrast medium employed and the survival time*

Survival time	Myelography with 0.4 ml Conray Meglumine 30					Myelography with 0.4 ml MP 2032 35				
	<1 day	1 day	7 days	3 weeks	6 weeks	<1 day	1 day	7 days	3 weeks	6 weeks
Cell infiltration in meninges	1	3	2	0	0	0	3	3	1	1
Cell infiltration in nerve root	1	1	0	0	0	0	1	0	0	0
Cell infiltration in spinal cord	1	0	2	1	0	0	1	1	1	0
Myelin degeneration	0	0	1	0	0	0	0	0	0	1
No. of rabbits with pathologic findings	2	3	3	1	0	0	3	3	1	1
Total No. of rabbits	4	5	8	6	5	2	1	1	8	6

filtration comprised mononuclear (Fig. 3 a) and polymorphonuclear cells with varying dominance. After Conray myelography the cells were predominantly polymorphonuclear. In the Conray group infiltration of the nerve roots was by polymorph cells in one instance in association with diffuse haemorrhage in the cord and meninges resulting from a fracture of the spine. Both mononuclear and polymorphonuclear cells were present in infiltration of the nerve roots after MP 2032 myelography. Two types of accumulations of cells localized to both white and grey matter were evident in the spinal cord. The first took the form of dark cells in the perivascular regions (Fig. 3 b) these were assumed to be leucocytes. The second consisted of small groups of somewhat paler cells which were not related to the blood vessels, and which were assumed to be glial cells. Myelin sheath degeneration in a nerve root (Fig. 4 a) was demonstrated in one case. Two rabbits have been excluded from the MP 2032 group in Table 4. The first was found dead on the ninth day after myelography, infiltration of mononuclear cells into the meninges and the perivascular areas of the cord were present. It was impossible to obtain a satisfactory preparation of the cord in the second rabbit.

*Einarson's galloxyamine* enabled a difference to be demonstrated in the stainability of the Nissl substance in the anterior horn cells of those rabbits that were killed in a tranquil phase after the myelography as compared with those that died during the myelography with muscle rigidity or convulsions. The first had in general chromoneutral to hyperchromic cells (Fig. 5), corresponding to a normal



Fig 3 a) Mononuclear cell infiltration in the pia mater of a rabbit 3 weeks after myelography with 0.4 ml MP 2032/35. To the left dura with arachnoid b) Perivascular cell infiltration in region of central canal in the same animal. To the left ependymal cells in central canal. Haematoxylin eosin  $\times 600$

state of gentle or initial activity while the latter had chromophobic cells (Fig 6), corresponding to a state of vigorous and prolonged activity (LORENTZEN 1950). However, no irreversible changes were ever observed in the larger neurons.

#### Discussion

Both Conray Meglumine and MP 2032 induce convulsions when injected into the subarachnoid space by suboccipital puncture, but to different degrees. Thus it was found that the interval between injection and the onset of convulsions was considerably shorter with Conray Meglumine than with MP 2032. While



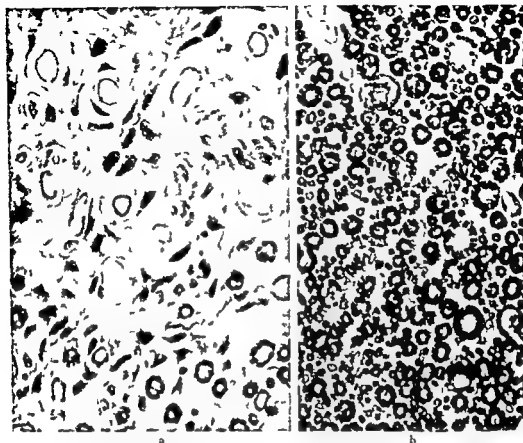


Fig. 4. Cross sections of degenerated (a) and normal (b) nerve root of rabbit 6 weeks after myelography with 0.4 ml MP 2032 35 °C. A few normal myelin sheaths seen at the bottom in (a). Luxol Fast Blue  $\times 600$ .

the dose of anaesthetic per unit time necessary to bring the rabbit through the period of convulsions followed a normal distribution and was independent of the contrast medium the mean dose was dependent on which contrast medium was employed, the dose necessary after Conray Meglumine was greater than that after MP 2032. Similarly, it was observed that the duration of anaesthesia followed a normal distribution with a variance that was independent of the contrast medium. The mean value depended somewhat on the contrast medium but the difference was less clearly demonstrated than the difference in the means of the dosage again the mean was higher for Conray Meglumine.

Myelography was followed by a transient increase in the protein content of the cerebrospinal fluid which was independent of which of the contrast media was used. There was also a rise in the leucocyte count in the cerebrospinal fluid.

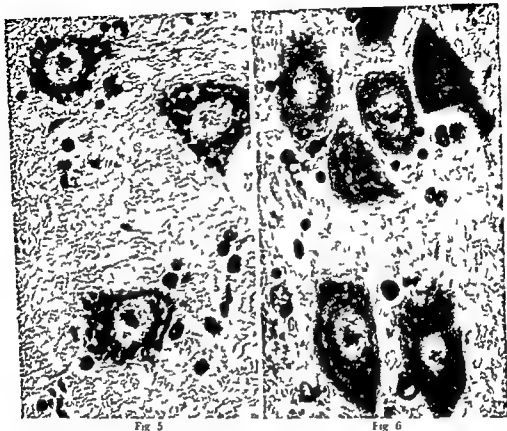


Fig 5

Fig 6

Fig 5 Hyperchromatic anterior horn cells from segment of L7 of a control rabbit killed at rest Einarson's gallocyanine stain  $\times 550$

Fig 6 Chromophobic anterior horn cells from segment of L7 of a rabbit that died with severe muscular rigidity 10 hours after myelography with 0.4 ml Contrav Meglumine 30% Einarson's gallocyanine  $\times 550$

but again there was no demonstrable dependence on the contrast medium employed

A relatively large group of control animals was employed in the evaluation of the histologic findings in the spinal cord nerve roots and meninges after myelography, this was mainly in order to determine whether there was any evidence of infectious disease in the central nervous system of the group of experimental animals used (LORENTZEN 1950 PETRI 1969). The findings were normal in all those rabbits that had not been subjected to any operative procedure. Abnormal findings were however recorded in 2 of the 8 rabbits in which cisternal puncture had been performed without the injection of contrast medium. This might suggest that it was not the contrast medium alone that was responsible

for the changes listed in Table 4, experiments in their elucidation are at present in progress. Certain differences in the histologic appearances following injection of the two contrast media were evident. Changes were present in 32 per cent of the animals that had received Conray Meglumine and 27 per cent of those that had had MP 2032. As such factors as severity and extent are involved, these figures have not been submitted to statistical analysis. The pathologic signs following Conray Meglumine give the impression of being more acute than those after MP 2032. One case had changes persisting as late as six weeks after myelography as one rabbit had nerve root degeneration following myelography with MP 2032.

### Acknowledgements

The author takes this opportunity of thanking Mrs Susanne Møller who carried out the statistical evaluations and Mallinckrodt Chemical Works USA and Astra Gruppen A/S Denmark who placed the contrast media at his disposal.

### SUMMARY

Conray Meglumine and MP 2032 (dimerised Conray Meglumine) have been used in myelography in rabbits. The relative suitability of the two contrast media was evaluated from the associated convulsions, protein concentration and leucocyte count in the cerebrospinal fluid as well as the histologic findings. Histology revealed a tendency for changes to be more frequent and more acute following the injection of Conray Meglumine.

### ZUSAMMENFASSUNG

Conray Meglumine und MP 2032 (dimersiertes Conray Meglumine) wurde für Myelographien bei Kaninchen verwendet. Die relative Verwendbarkeit der beiden Kontrastmittel wurde nach den begleitenden Krämpfen, der Proteinkonzentration und der Leukozytenzahl in der cerebrospinalen Flüssigkeit sowie den histologischen Befunden beurteilt. Die Histologie zeigt eine Tendenz für häufigere und mehr akute Änderungen nach der Injektion von Conray Meglumine erkennen.

### RÉSUMÉ

L'auteur a utilisé pour la myélographie chez des lapins le Conray Meglumine et le MP 2032 (dimère du Conray Meglumine). La tolérance de ces deux moyens de contraste a été appréciée d'après les convulsions, la concentration en protéine et la numération des leucocytes dans le liquide céphalo rachidien ainsi que d'après les constatations histologiques. L'histologie a montré que les lésions ont tendance à être plus fréquentes et plus aiguës après l'injection de Conray Meglumine.

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## COMPLICATIONS OF MYELOGRAPHY WITH CONRAY MEGLUMIN

by

J PRÆSTHOLM and J LESTER

Conray Meglumin has been employed in lumbar myelography without spinal anaesthetic since 1964 (CAMPBELL *et coll*) The results of 847 myelographies carried out with Contrix (= Conray Meglumin 282) were discussed at a symposium in Paris in October 1968 (SERRE 1968) and included a patient with a fatal course associated with myoclonia Another fatality has recently been mentioned by IRSTAM *et coll* (1970) The present authors in an earlier report (PRÆSTHOLM & LESTER 1970) described the results of 136 Conray myelographies with due regard to the quality of the myelogram, the correlation between the radiologic and operative findings, and the complications The purpose of the present report was to analyse the occurrence of side effects in an extended material

*Material and Technique* The material comprised 318 myelographic examinations 223 of which were carried out on men and 95 on women 58 were under 30 years of age, 123 between 30 and 44, 116 between 45 and 60 years and 21 patients were 60 or over

Premedication consisted of Valium by mouth on the evening before the investigation and 7.5 mg Valium intramuscularly 45 minutes before the investiga-

tion. The second dose of Valium in the first 136 patients was 10 mg and was administered in the department of neuroradiology immediately before the myelography, it was necessary to alter this part of the premedication because of a tendency to hypotension when the patients were placed in the sitting position.

The dose of medium was 5 ml Conray Meglumin 282 diluted with 3 ml CSF in 281 patients and 6 ml Conray Meglumin 282 diluted with 2 ml CSF in 31 patients. For special reasons 4 patients received 4 ml and 2 patients 7 ml Conray Meglumin 282.

The first 17 patients lay in bed with the head-end elevated at an angle of 15° for 24 hours after the investigation. The last 301 patients were put to bed in the sitting position for 6 hours, and thereafter lay with the head end raised until the following morning.

The blood pressure was registered before, during and for at least 4 hours after myelography. The rectal temperature was usually also taken and a leukocyte count performed. Special observation charts were filled in for all patients.

## Results

*Side effects during myelography.* A total of 11 patients had slight symptoms. Three complained of an accentuation of their usual pain, six of lumbar pain of a new type, and one of pain radiating down one leg. One patient developed paraesthesia of the legs after 15 minutes. Myelography was carried out twice in this patient, and on each occasion he developed paraesthesia followed about two hours later by clonic spasm of the legs. In 19 patients (6 per cent) a transient fall in blood pressure of more than 20 mm Hg occurred immediately after the injection of the contrast medium. Only one patient received treatment; this took the form of a single intravenous injection of 50 mg ephedrine.

*Side effects after myelography.* The most prominent side effects after Conray myelography were irritative phenomena in the form of clonic and tonic spasm of the legs. The phenomena in the 318 myelographic examinations are recorded below.

	Cases
Clonic muscle spasm of legs requiring treatment	12 (3.8 %)
Clonic muscle spasm of legs not requiring treatment	5 (1.6 %)
Tonic muscle spasm of legs	11 (3.5 %)
Pain in lumbar region	11 (3.5 %)
Pain in legs	5 (1.6 %)
Total	44 (13.8 %)

Twelve patients developed painful clonic spasm of the legs after a latent period of 1.5 to 5 hours. These consisted of rapid twitching in the legs lasting for a few seconds at intervals of 10 seconds when the attack was at its height. Irritation of any description might provoke a new attack. Apart from this the reflexes in the legs were unaffected and there was no flexor plantar response. During the attack the patients perpired heavily and became extremely exhausted when the attacks were of long duration. No fall in blood pressure ever occurred and there was no loss of consciousness or generalized convulsions. It was possible to abolish the attack by the intravenous injection of Valium, but it was sometimes necessary to administer a high dose i.e. of up to 40 mg over the course of 15 minutes. One patient was treated for muscle spasm 16 hours after the myelography. A further 5 patients stated that they had had a series of spasms of the legs, but these were not painful and disappeared without treatment. The myoclonia was followed by a tight painful feeling in the muscles of the legs in 5 patients. Eleven patients had tonic spasm of the legs without myoclonia. A few of these patients were treated with intramuscular injections of Valium. Sixteen patients complained only of pain in the loins and legs after the myelography. The same symptom was registered in a number of the patients who later developed clonic or tonic muscle spasm.

The most common symptom after Conray myelography was headache, which occurred in 89 patients (28 per cent). It was usually light. Thirteen patients still suffered from headache 48 hours after the myelography. The pain lasted for 8 to 10 days in 3 patients and in one of them it was associated with rigidity of the neck and back. At no time was the temperature in this patient raised. A fall in systolic blood pressure from 120 to 80 mm Hg was observed in 2 patients but returned to the initial level without treatment. Nine patients experienced nausea and vomiting. Three patients had retention of urine for 24 hours following the myelography. One patient had transient loss of power in the right leg, this patient already suffered from pain radiating into the right leg associated with right sided prolapse of the third lumbar intervertebral disc. One patient complained of diplopia, one of tinnitus and 3 of dizziness. No other sequelae occurred.

Fifty-seven of the 300 patients in whom the rectal temperature was recorded had light hyperpyrexia on one or more occasions during the first three days after the myelography. The maximum increase over a morning temperature of 37° C and an evening temperature of 37.5° C was raised by 0.8° C in one patient. The leukocyte count was recorded on the third day after the myelography in 189 patients, 3 of whom had a slight leukocytosis. The ESR was measured in 196 patients, 16 of whom had an increase from normal levels to those at the lower end of the pathologic range. Of the patients with leukocytosis and increased ESR after the myelography, 2 suffered from myoclonia, one from tonic spasm of the leg, 4 from headache and 5 patients had a slight increase in temperature.

### Discussion

The most obvious advantage of Conray Meglumine in lumbar myelography is its low tendency to cause direct irritation which in contrast to Methiodal myelography (AHLGREN & PRESTHOLM 1969), renders the employment of local anaesthesia superfluous. The subsequent simple and uncomplicated examination procedure without the threat of a fall in blood pressure is a considerable improvement for both patient and investigator although the clonic and tonic spasms of the legs which occur as a relatively late sequela have proved to be a marked disadvantage. In all patients with such spasms it was possible to observe the contrast medium relatively high in the spinal canal in the myelograms demonstrating the conus medullaris. Contrast medium in the thoracic region of the spinal canal was about twice as common in patients with spasm as in those who did not experience this side effect. Corresponding to this myoclonia occurred particularly in patients who had received a large dose of contrast medium or in whom there was total or partial blockage of the spinal canal by a large prolapse below the level of the injection.

Pain in the loin often preceded the development of myoclonia and it is possible that Valium would perhaps have prevented the development of muscle spasm. Immediate intravenous administration of Valium in adequate doses at the onset of the myoclonia of the legs appears to inhibit its further development.

The authors kept 301 of the patients included in the material in the sitting position after the myelography although it was occasionally rather difficult partly due to a disc lesion and partly to headache. It is probable that the sitting position after the lumbar puncture increases the leakage of CSF into the peridural space and thereby leads to an increase in the incidence of headache.

Precautions which would tend to prevent the development of muscle spasm would thus include

- (1) Use of the smallest possible dose of contrast medium. Injection during fluoroscopy will afford a guide as to the necessary amount.
- (2) Maintenance of the sitting position after the myelography as the heavy contrast medium runs downwards.
- (3) Evacuation of cerebrospinal fluid containing contrast medium where the column of medium has reached a high level e.g. in connection with a narrow sacralis or a spinal block caused by a large prolapse.
- (4) Finally, the prophylactic administration of Valium would probably inhibit the development of myoclonia.

Since both DAVIS *et al.* (1968) and IRSTAM *et al.* (1970) have warned against Conray myelography in patients with epileptic seizures there must be considered as absolute contraindications to this kind of myelography



### Conclusion

Conray Meglumin may be injected into the lumbar subarachnoid space without any immediate discomfort to the patient and in contrast to Abrodil, requires no spinal anaesthetic. However a considerable complication of the investigation is the relatively late occurrence of irritative phenomena related to a high level of contrast medium in the myelogram and probably due to a direct influence on the spinal cord. Low doses of contrast medium, keeping the patient in the sitting position after the myelography, and evacuation of cerebrospinal fluid and medium when the latter is at a particularly high level bring this sequela down to an acceptable minimum. Intravenously injected Valium has never failed to control the myoclonia.

### SUMMARY

The complications of 318 lumbar myelographies with Conray Meglumin are analysed. The most prominent side effect was clonic spasms of the legs and occurred in 12 patients. The measures to keep untoward sequelae to a minimum are considered.

### ZUSAMMENFASSUNG

Die Komplikationen bei 318 lumbalen Myelographien mit Conray Meglumin werden analysiert. Der ausgeprägteste Nebeneffekt war clonische Spasmen der Beine. Diese traten bei 12 Patienten auf. Die Massnahmen die nachteiligen Folgen auf ein Minimum einzuschränken werden diskutiert.

### RÉSUMÉ

Les auteurs analysent les complications de 318 sacroradiculographies lombaires faites avec Conray Meglumin. L'incident le plus important a été les spasmes cloniques des membres inférieurs qui se sont produits chez 12 malades. Les auteurs examinent les mesures destinées à limiter au minimum les incidents.

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## BLADDER FUNCTION DURING LUMBAR MYELOGRAPHY

An experimental investigation in cats

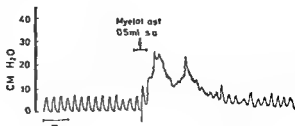
by

I O SKALPE and R GJØNE

All the present myelographic contrast media have obvious shortcomings. Sodium iodomethanesulfonate (Myelotrast Kontrast U Abrodil) is most commonly used in Scandinavia. The irritating effects of this agent demand spinal anesthesia and its application is confined to the lumbar subarachnoid space. Meglumine isothalamate (Conray Meglumine) was introduced both for ventriculography and myelography by CAMPBELL et coll (1964). This medium may be considered superior to Myelotrast in so far as the density is better and the irritation of the leptomeninges less (AHLGREN 1969). It may therefore be used without spinal anesthesia. It should be stressed however, that the complication frequency with Myelotrast myelography is low (AMUNDSEN et coll 1963, KOLSTAD & SOLEH 1959). Complications although usually slight and transitory seem to be more common with Conray (CAMPBELL et coll 1964, DAVIES et coll 1968, LIGRE et coll 1968).

Three water soluble contrast media have been investigated: Myelotrast 20 % (107 mg I/ml), Conray Meglumine (282 mg I/ml) and Isopaque Cerebral (280 mg I/ml). Isopaque Cerebral (meglumine metrizoate) has a chemical formula similar to Conray Meglumine and competes favourably with the latter in cerebral

Fig. 1 Continuous intravesical pressure recording in male cat illustrating the effect of an injection of Myelotest into the lumbar subarachnoid space. The injection is immediately followed by a marked rise in bladder pressure indicating sustained vesical contraction which resulted in micturition.



angiography (LÅNDERVOLD & ØNGRE 1967). It has not previously been used in myelography.

The evaluation of the irritative effects was based upon the influence of contrast media on bladder motility observed by continuous cystometric recording. The cystometric examination elucidates autonomic effects exerted by irritation of the nerve supply to the bladder. In addition motorial responses from the hindlimbs and tail were noted. The density produced by the three contrast media was compared and their absorption rates were estimated from the roentgenograms.

**Material and Methods.** Twelve cats, mostly males (weight 2.0 to 6.5 kg) were used. The anaesthesia was initiated with ether followed by pentobarbital sodium (Nembutal) 25 mg/kg intraperitoneally; additional doses of 10 mg/kg were given when necessary. With two exceptions a relatively light and steady anaesthesia was obtained.

The investigation of bladder motility was based upon the direct cystometry method introduced by GJØNE & SETEKLEIV (1963). Cystostomy was performed through a midline suprapubic incision and a catheter was inserted into the fundus region and sutured to the bladder wall. Continuous intravesical pressure recording was established by means of a Statham pressure transducer connected to a Philips recorder. The bladder was about half filled with 0.9% saline. Partial laminectomy was performed in the upper lumbar region and the dura was opened by puncture with a cannula; a catheter (Portex No. B 205, outer diameter 0.75 mm) of approximately the same diameter as that of the cannula was then introduced caudally into the subarachnoid space until the catheter tip reached the level of L<sub>5</sub>–L<sub>6</sub>. The animal was placed on the right side on the roentgen couch and the anterior part of the body was elevated to an angle of 10°. Roentgenograms (vertical beam direction, 0.06 to 0.1 s, 62 to 66 kV, 100 mA) ensured the correct position of the contrast-filled catheter. Films were also obtained immediately after the injections and 15, 30, 45 and 60 minutes later. All injections through the catheter were made slowly over 30 seconds in order not to cause undue increase in the CSF pressure.

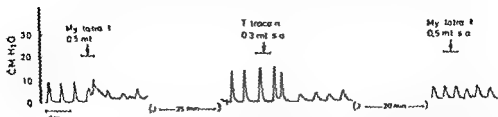


Fig 2 Effects of spinal anesthesia with 0.3 ml 0.5 % Tetracain on bladder motility. Marked reduction in the spontaneous rhythmic contractions. Myelotrast injected during spinal anesthesia did not influence the intravesical pressure recording

## Results

**Cystometric investigation** Regular spontaneous contractions were recorded for at least half an hour before the experimental procedure was initiated. All the changes in the bladder motility described below occurred during or immediately after the contrast medium injection.

Six cats served as controls and received 0.5 ml 0.9 % saline through the subarachnoid catheter. In 4 cats saline was given as the first injection and in 2 cats 0.5 ml Conray had been administered two hours before the saline. No changes in the bladder motility were evident.

All cats had 0.5 ml Myelotrast. Myelotrast was given as the first injection in 2 cats and as the second contrast medium injection in 10 animals. Characteristic alterations in the cystometrogram were elicited immediately after the injection of Myelotrast in all cats. An increase in the intravesical pressure occurred accompanied by micturition either complete (Fig 1) or dropwise with incomplete bladder emptying (Fig 2).

In 3 cats 0.3 ml Tetracain was administered through the subarachnoid catheter. The amplitudes of the vesical contractions were immediately reduced (Fig 2). A subsequent injection of Myelotrast had no further effect upon the spontaneous bladder activity.

Six cats were given Conray Meglumine 0.5 ml subarachnoidally. In 5 of these as the first contrast medium injection. In one cat 0.5 ml Myelotrast had been administered 15 minutes before the Conray injection. In this animal a moderate rise in the intravesical pressure followed by increased frequency of the spontaneous contractions was observed (Fig 3). A slight reduction in the spontaneous contractions occurred in one cat and they were completely abolished for about five minutes in another cat. No changes occurred in the rest of the animals.

Five cats initially received 0.5 ml Isopaque Cerebral subarachnoidally. The effect upon the cystometrogram was principally the same as that produced by Conray Meglumine. A moderate rise in the intravesical pressure and increased

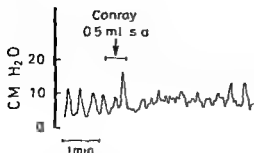


Fig 3

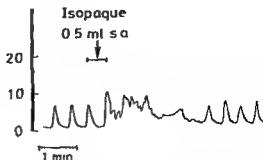


Fig 4

Fig 3 Effect on rhythmic bladder activity caused by injection of Conray Meglumine into the lumbar subarachnoid space. A moderate rise in intravesical pressure represented by a peak is followed by increased frequency of the rhythmic contractions.

Fig 4 Isopaque Cerebral injected in the lumbar subarachnoid space. The influence on bladder motility as observed with Conray Meglumine is about the same.

frequency of the spontaneous contractions were recorded in one cat (Fig 4). Reduction in the amplitudes of the spontaneous contractions occurred in another. No changes were observed in the rest of the animals.

The catheter was accidentally inserted into the epidural space in one cat, in this position injection of Myelotrast and Conray Meglumine failed to influence bladder activity. The catheter was then introduced into the subarachnoid space and following further injections of contrast media characteristic changes in vesical motility occurred.

**Somatomotorial effects.** Myelotrast caused in all but 2 cats tonic extensor spasms of the hindlimbs and tail lasting a few seconds followed by clonic spasms of 1 to 2 minutes duration. No spasms occurred in the abdominal muscles and the changes in the cystometrograms were evidently independent of the spastic movements of the hindlimbs. Marked hyperirritability of the hindlimbs and tail were evident during the next few hours. Clonic spasms were elicited even by a slight touch, spinal anesthesia immediately abolished these somatomotorial responses. The narcosis was deeper than in the other animals in 2 cats in which no somatomotorial responses were evident as an additional dose of Nembutal had been given during the operation for pain reactions.

In 4 of the 6 cats receiving Conray Meglumine and in 3 of 5 cats having Isopaque Cerebral, similar somatomotorial responses as provoked by Myelotrast occurred. The reactions were however more moderate with these media. No somatomotorial responses were observed in 4 cats, changes in the cystometrograms were evident in 2 of these animals.



Fig 5 Lumbar myelography with a) 0.5 ml Myelotrust b) 0.5 ml Conray Meglumine and c) 0.5 ml Isopaque Cerebral. Poor density with Myelotrust but satisfactory with Conray Meglumine and Isopaque Cerebral

*Roentgenologic investigation* Filling of the lumbar subarachnoid space with Myelotrust was poor. No contrast medium was usually observed in the films and only in a few instances was slight filling evident (Fig 5 a).

The filling and density with Conray Meglumine and Isopaque Cerebral were good with no difference between the two media (Fig 5 b and c). The contrast column could be followed to the upper lumbar region. Both media were resorbed in about an hour and had usually disappeared at 45 minutes.

### Discussion

Little attention has been paid to bladder function during lumbar myelography in spite of the fact that parasympathetic (motor) influence on bladder motility is mediated through the sacral nerve roots. FISHER (1965) observed that several cats lost urine and feces during experimental Conray Meglumine myelography but

no other investigation of bladder activity during myelography appears to have been published

The present investigation indicates that the introduction of a contrast medium into the lumbar subarachnoid space may cause irritation of the nerve supply to the bladder resulting in changes in the cystogram, as described Myelotrast which from experimental and clinical experience is known to be highly irritating (FUNKQUIST & ØREL 1960, KOLSTAD & SOLEV 1959), may produce profound changes in the cystometrogram. Conray Meglumin and Isopaque Cerebral also affect bladder motility in some animals but the changes are less marked than with Myelotrast. Six of the animals in which no somatomotorial response was observed had irritative effects from the contrast media as revealed by the cystometrography. Intravesical pressure recording may therefore be expected to serve as a guide in the evaluation of new myelographic contrast media.

Myelotrast is far from being an ideal contrast medium. This is confirmed in the present investigation by the marked cystometrographic changes, the violent somatomotorial reaction and the poor quality of the films. Both Conray Meglumin and Isopaque Cerebral seem to be better suited for myelography although these media also possess local irritating properties. It is obvious that the search for new and better myelographic contrast media should be intensified.

## SUMMARY

Lumbar myelography has been performed in 12 cats with Myelotrast, Conray Meglumin and Isopaque Cerebral. Direct cystometry indicated that Myelotrast caused bladder contraction followed by micturition in all animals. Conray Meglumin as well as Isopaque Cerebral sometimes produced changes in bladder activity. The investigation revealed that cystometry may be used to reveal the effects of irritation caused by myelographic contrast media.

## ZUSAMMENFASSUNG

Eine lumbare Myelographie wurde bei 12 Katzen mit Myelotrast, Conray Meglumin und Isopaque Cerebral durchgeführt. Die direkte Blasendruckmessung zeigte, dass Myelotrast bei allen Tieren eine Kontraktion der Blase mit nachfolgender Miktions zur Folge hat. Conray Meglumin sowie Isopaque Cerebral rufen gelegentlich Veränderungen der Blasenaktivität hervor. Aus der Untersuchung geht hervor, dass Blasendruckmessungen verwendet werden können, um Irritationseffekte, die durch myelographische Kontrastmittel hervorgerufen werden, festzustellen.

## RÉSUMÉ

Les auteurs ont fait sur 12 chats une myelographie lombaire avec le Myelotrast, le Conray Meglumin et l'Isopaque Cerebral. La cystometrie directe montre que le Myelotrast provoque une contraction vésicale suivie de mictions chez tous les animaux. Le Conray Meglumin et

l'Isopaque Cerebral produisent dans certains cas des modifications de l'activité de la vessie. Ce travail de recherche a montré qu'on peut utiliser la cystométrie pour mettre en évidence les effets d'irritation causés par les moyens de contraste myélographiques.

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## ANALYSE ISODENSITOMETRIQUE DES RADIOGRAPHIES

par

J P BRAUN, J BLANC et A WACKENHEIM

L'amélioration photographique de l'information de l'image radiographique proposée de deux techniques : la copie de la radiographie qui modifie le contraste de l'image et la technique d'extraction d'information déterminée soit par sous-traction soit par analyse densitométrique. Cette dernière technique a été peu utilisée à cause des travaux photographiques compliqués qu'elle nécessite et du grand nombre d'artefacts produits. L'utilisation du film Agfacontour simplifie considérablement le travail et évite tout artefact de ce genre. Grâce à ce film nous avons repris l'étude densitographique des clichés radiographiques sans parler de son application en thérapie pour l'établissement rapide des courbes isodenses.

Ce film comporte deux émulsions dont les courbes caractéristiques de noircissement sont inverses. En d'autres termes, les deux émulsions ont superposées l'une positive, comme celle d'un film contretype l'autre négative analogue au film radiographique. La courbe positive se déplace sur l'axe des abscisses vers la courbe négative suivant la qualité spectrale de la lumière utilisée. À cet effet on emploie pour ce film des filtres jaunes de densité croissante. Les deux courbes forment une figure en « U » majuscule dont les branches sont d'autant plus rapprochées que le filtre jaune est dense. Le point de croisement à la base des deux courbes correspond à une valeur d'éclairement précise qui est transcrite par une faible densité (par ex. le filtre n°3 équivaut à D07 et sans filtre équivaut à D01). Le déplacement de l'ensemble des courbes dépend uniquement de la durée de l'exposition. Nous constatons que cette émulsion est en

Fig 1 Courbes de noircissement En ordonnée on a représenté la densité et en abscisse le logarithme de l'énergie utilisée La courbe positive (gauche) se place sur l'axe des abscisses vers la courbe négative (droite) grâce à l'utilisation des filtres jaunes de densité croissante Les deux courbes forment une figure en

« U » dont les branches sont d'autant plus rapprochées que le filtre jaune utilisé est dense Le point de croisement à la base de deux courbes correspond à la valeur d'éclaircissement précise transcrite par une faible densité

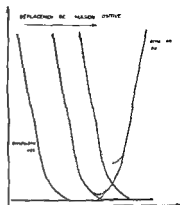
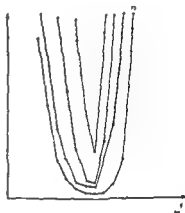


Fig 2 Courbes de noircissement utilisant différents filtres Par exemple sans filtre F 0 équivalent à D 0 1 (niveau du voile) avec filtre n 1 équivalent à D 0 avec filtre n 2 équivalent à D 0 3 et avec filtre n 3 équivalent à D 0 7 Le déplacement de l'ensemble de ces courbes dépend de la durée de l'exposition Ces courbes indiquent les valeurs réelles de la densité mesurée et montrent l'effet du contraste Rien n'est retenu que l'intervalle des densités des copies par le filtre d'où le terme de puits



quelque sorte un puits, un entonnoir à pente raide isolant toutes les zones d'un image de même densité en les traduisant par un voile gris faible identique. On conçoit immédiatement son intérêt pratique qui permet par simple copie contact d'obtenir une carte de répartition de la densité.

Nous vous rappelons que le faisceau des rayons de roentgen qui émerge de l'objet radiographié n'est pas uniforme du point de vue qualitatif et quantitatif. On peut le représenter par des tiges de longueur proportionnelle soit à l'énergie soit à l'intensité du rayonnement. C'est la représentation de Spiegler qui met ainsi en évidence le relief du faisceau.

L'écran fluorescent donne en chaque point une intensité lumineuse proportionnelle au nombre de photons absorbés et le récepteur photographique fait de

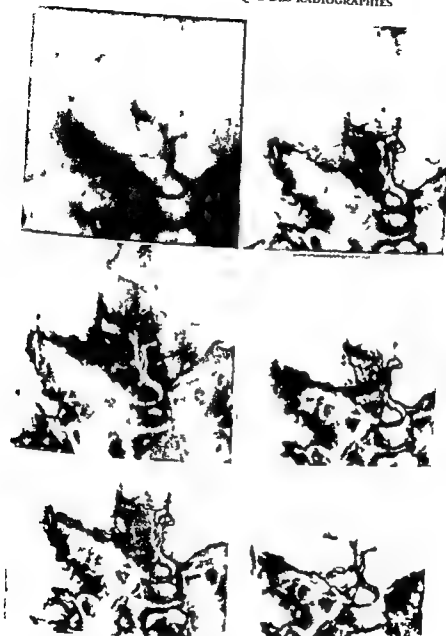


Fig 3 Angiographie vertébrale droite dans un ca. de neurinome de l'angle ponto-cérébelleux. La radiographie standard montre le déplacement vasculaire des branches de l'artère cérébelleuse postéro-inférieure droite. L'extraction d'images par la méthode d'isodensitométrie montre sur différents niveaux les altérations topographiques et l'opacification tumorale.

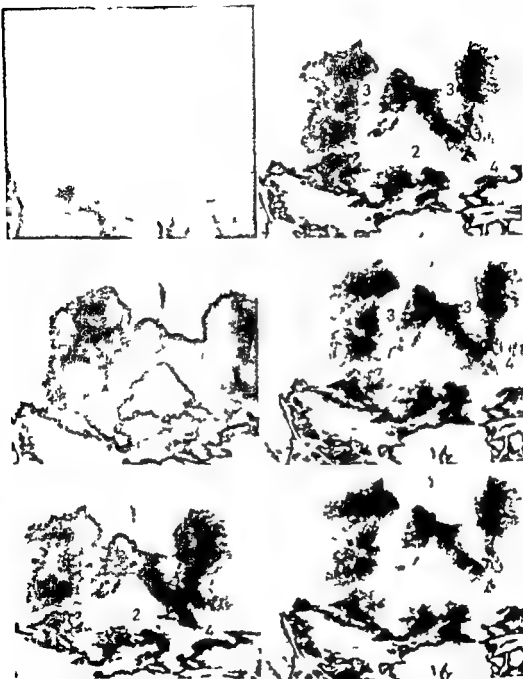


FIG. 4. Encéphalographie gazeuse du neurinome du nerf acoustique. L'image d'encéphalographie standard montre l'altération de la cistère latéro-pontique par le neurinome. Les différentes incidences faites par la technique radiodensitométrique permettent d'isoler nettement le noyau tumoral de l'angle ponto-cérébelleux droit qui est individualisé grâce à cette technique. Le 4<sup>ème</sup> ventricule et les cisternes ambiantes sont visibles ainsi que l'angle ponto-cérébelleux gauche. 1. Neurinome. 2. 4<sup>ème</sup> ventricule. 3. Cisternes ambiantes. 4. Angle ponto-cérébelleux gauche.

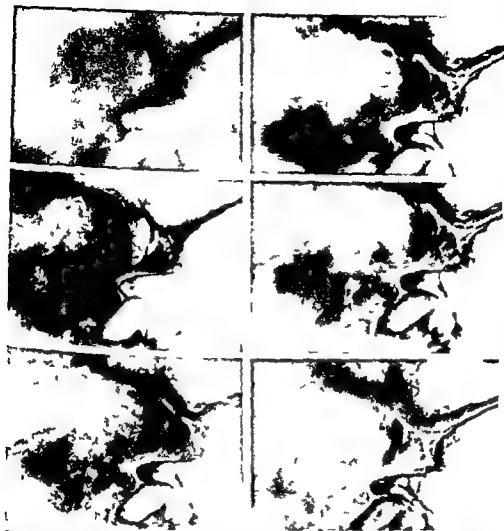


Fig. 5. Méningiome de la petite aile du sphen. Pendant la phase uérotemporale on remarque une petite opacité arrondie dans la projection phéno-temporale. Cette opacité apparaît bien délimitée sur les différentes copies isodensitométriques réalisées à partir du cliché standard.

rième en donnant une quantité d'argent proportionnelle après le développement aux photons lumineux absorbés. C'est ainsi que le relief est devenu un plan.

Le film Agfacontour permet cette analyse verticale des courbes isodenses analogues aux courbes de niveau qui représente sur un plan les variations d'un troisième paramètre : la valeur de la transmission spatiale de l'objet représentée par la densité.

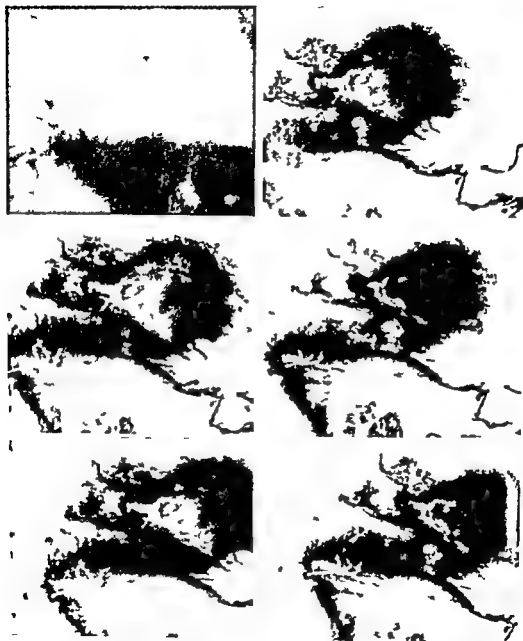


Fig 6 Microangiographie vertébrale dans une tumeur du tronc cérébral. L'incidence standard (à gauche) montre l'altération des veines ponto-mésencéphaliques et basales. Les coupes séquentielles montrent les vaisseaux à différents niveaux et permettent de différencier les affluents veineux de calibre très fin.

Nous avons l'habitude de voir l'image radiographique sous deux dimensions en nous référant à nos connaissances médicales pour reconnaître la structure et pour en estimer l'opacité radiologique. Notre œil en effet est incapable de donner une valeur à une densité bien qu'il soit très sensible à des faibles variations de celle-ci.

Nous pensons qu'une orientation nouvelle est possible par l'établissement de ces cartes d'isodensité que le procédé d'isofrontage permet d'établir rapidement. L'utilisation d'un puits plus ouvert peut mettre à profit le contraste des deux courbes autour de la bande d'isodensité. Nous pouvons ainsi voir les détails de la structure de l'organe avec une grande précision. Il faut signaler que les densités de valeur inférieure au point d'isodensité ont traités sur la courbe positive et les densités de valeur supérieure sur la courbe négative. La référence à la radiographie de base est absolument nécessaire car les images partielles qu'on obtient ont essentiellement des compléments des verniers.

Les différents temps de pose nécessaires pour déplacer la structure de la pente positive à la pente négative nous valent des plans successifs. C'est pour cette raison que ce procédé a été appelé la tomographie de la radiographie. Cette technique ne possède donc pas d'effet distrayant car l'extraction n'est pas faite par élimination mais par isolement. Nous avons illustré cette première étude par plusieurs exemples du domaine neuroradiologique.

## RÉSUMÉ

L'isodensitométrie est une analyse verticale du cliché radiographique par extraction des courbes isodenses qui reproduisent les densités en négatif et positif sous forme de lignes. Ses contours s'étalent sous forme de surfaces et sont appelés équidensités. Le film utilisé possède une émulsion spéciale dont la courbe caractéristique de noircissement a la forme d'un « U » dont l'écartement peut être modifié au moyen d'un filtre. Par cette technique de nombreuses informations radiologiques invisibles sur le cliché standard sont mises en évidence.

## SUMMARY

Isodensitometry consists in an analysis of the radiograph in depth points of the same negative and positive density being plotted to form curves of isodensity. A special type of film with a U-shaped density curve is employed and with suitable filters facilitates the separation of density values. The new method reveals objects not evident in the orthodox film.



## ZUSAMMENFASSUNG

Die Isodensitometrie ist eine Tiefenanalyse des Röntgenbildes, die durch das Anlegen von Isodichten Kurven, die die gleichen positiven oder negativen Halbtonwerte verbinden, ermöglicht wird. Ein Film mit einer Spezialemulsion ist notwendig, dessen Schwarzungskurve eine charakteristische U-Form besitzt und die es ermöglicht, mit Hilfe von Filtern eine grössere Trennung der Schwarzungsgebiete zu erzielen. Mit dieser neuen Methode ist es möglich, auf dem gewöhnlichen Film unsichtbare Objekte sichtbar zu machen.

## POSITIONING FOR POSITIVE CONTRAST CISTERNOGRAPHY

by

PAUL M. DUCHESNEAU

Early in the development of positive contrast (Pantopaque) cisternography many clinicians were content with fluoroscopic spot films taken with the patient prone and his head rotated into nearly a Steevens position. The Pantopaque was allowed to trickle into the internal auditory canal (Fig 1 a) with a greater or lesser amount layered on the interior surface of the cerebellopontine angle. This latter portion of Pantopaque extending along the posterior surface of the petrous pyramid posterior to the internal auditory canal often obscured the contrast medium actually in the canal (Fig 1 b). This Steevens spot film is still used in many institutions often to the exclusion of any other films.

A second projection now used by many is a horizontal beam direction cross-table film with the patient's head in the same oblique projection. In this view, the Pantopaque in the internal auditory canal is dependent and seen as a finger like projection clear of the other Pantopaque in the cerebellopontine angle region (Fig 2).

After a little practice, most radiologists who perform this examination become familiar with the landmarks and proficient in interpretation of the radiographic findings in these two views. With a definite tumor (Fig 3) that produces a well defined cap in the Pantopaque orientation of the films and



Fig 1 a) Fluoroscopic spot film. Patient prone with head in Stevers position. Internal canal cell filled with Pantopaque but nearly impossible to delineate due to superimposed contrast medium in cerebellopontine angle. b) Base view. Pantopaque in internal auditory canal clearly shown separate from contrast medium behind it along posterior surface of petrous pyramid.

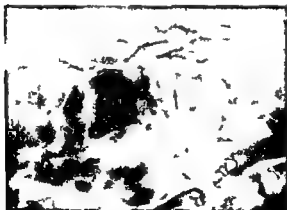


Fig 2 Cross-table horizontal beam direction. Head of patient still in Stevers position. Pantopaque hanging down in internal auditory canal.



Fig 3 Acoustic neuroma with Pantopaque column capping the medial border of a tumor. Fluoroscopic spot films and lateral decubitus ap or pa films usually suffice for adequate demonstration. a) Spot film in Stenvers position. b) Lateral decubitus position. Horizontal beam direction, pa view. c) Same position, half axial view.



Fig 4 Fluoroscopic spot films. After filling canal with Pantopaque it may be more easily seen if the cerebellopontine angle is partly emptied by tilting the head of the table upwards to 30 to 50°. a) Cerebellopontine angle filled with Pantopaque. b) Angle region partly emptied with internal canal more easily seen.

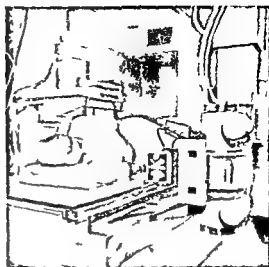


Fig 5 Patient position for Pantopaque cisternography. After filling one canal under fluoroscopic control and taking spot films in Stenvers position patient is turned on his side (lateral decubitus) with the filled canal dependent.

identification of the mass is no particular problem. With normal cisternograms though, or with minute tumors, this is not the case. When referring clinicians cannot evaluate the radiologic findings of the patients, the difficulty is obviously one of poor presentation of the information. Most physicians interested in the anatomic area in question can identify and evaluate the internal auditory canals.

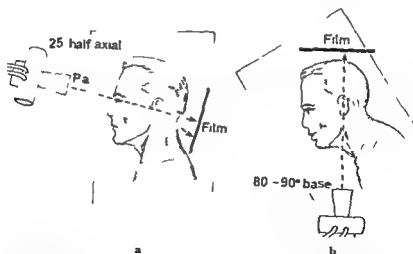


Fig 6 a) Path of central beam. Patient in lateral decubitus position using cross table horizontal beam direction. The head tube and film are aligned to produce p.a. and half axial views or a p. and reverse half axial. b) Base view is obtained by extension of the head and realignment of tube and film. Angles of 80° to 90° from the cantho-meatal line are satisfactory.

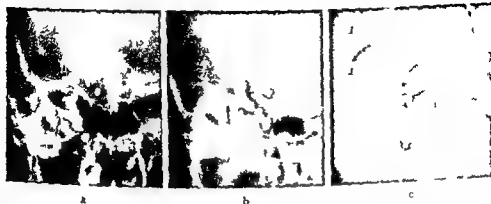


Fig 7 a) Pa view Pantopaque in internal auditory canal clearly demonstrated (normal) b) Half axial view c) Basal view Pantopaque filling large normal canal

on conventional roentgenograms of the skull. Four projections best demonstrate the canals. The first projection is a straight pa view with the central beam directed down the cantho meatal line so that the internal auditory canals are projected in the center of the orbit. The second projection used is a 25° half axial view. The canals are also clearly seen on a well penetrated basal view; they are also well demonstrated although foreshortened in Stenvers' projection. Since all clinicians, including otolaryngologists, neurologists and neurosurgeons as well as radiologists are accustomed to looking at these projections, it was considered helpful to use the same projections to demonstrate normal or abnormal anatomy when investigating this region with Pantopaque.

**Technique.** The cerebellopontine angle and internal auditory canal (Fig 4) are filled with Pantopaque in the usual manner. Only enough Pantopaque is used to fill the normal internal auditory canal and to place a thin layer along the dependent surface of the cerebellopontine angle to exclude other masses in this area. Spot films are then taken, in approximately the Stenvers' position. When the canal is normal, some attempt is made to empty the region of the cerebellopontine angle of Pantopaque by tilting the table partly upright while retaining the Pantopaque in the internal auditory canal.

The table is then leveled and the patient rotated directly onto his side in a lateral decubitus position (Fig 5). The head is made parallel to the table top with a horizontal folded sheet or towels. Roentgenograms are then taken with a horizontal roentgen beam direction to give pa (Fig 6 a), half axial and basal views (Fig 6 b). The patient's face is simply tilted toward the feet or up toward the top of the table. The tube and film are aligned in such a way that the

central beam takes the desired course with relation to the cantho-meatal line Fig 7 a shows the Pantopaque dependent in the internal auditory canal in the standard p a projection Fig 7 b the canal in the half axial projection and Fig 7 c the Pantopaque in a slightly enlarged internal auditory canal on the right side When satisfactory films are obtained on one side, the above procedure is repeated on the other side

### Conclusion

The above outline system of positioning the patient for positive contrast cisternography has been tried and found highly successful With familiar anatomy presented in familiar projections, normal or abnormal findings are easily recognized Another obvious advantage of these views is that the Pantopaque in the internal auditory canal is not obscured by adjacent contrast medium in the cerebellopontine angle

### SUMMARY

A system of patient positioning for positive contrast (Pantopaque) cisternography is described The internal auditory canal and cerebellopontine angle are filled and fluoroscopic spot films taken The patients is then rotated into lateral decubitus position Pa half axial and base views are taken with cross table horizontal beam direction

### ZUSAMMENFASSUNG

Ein System für die Lagerung von Patienten zur positiven Kontrast (Pantopaque) Cisternographie wird beschrieben Der innere Gehörkanal und der Kleinhirnbrückenwinkel werden gefüllt und während Durchleuchtung Augenblicksbilder gewonnen Der Patient wird dann in eine seitliche Decubitus Lage gedreht Danach werden halb axiale und Basisbilder hergestellt mit einem zum Kreuzzusch horizontalen Strahlengang

### RÉSUMÉ

Description d'un système de mise en place du malade pour cisternographie avec un moyen de contraste positif (Pantopaque) Le conduit auditif interne et l'angle ponto cérébelleux sont remplis et on prend des films au sélecteur sous contrôle radioscopique Puis le malade est tourné en decubitus latéral On prend des radiographies de face en incidence semi axiale et en incidence de la base du crâne avec un rayonnement horizontal perpendiculaire au grand axe de la table

## THE NEUROPHYSIOLOGIC ACTION OF CONTRAST MEDIA

by

R. L. GOSETTE

The contrast media employed in cerebral angiography are not biologically inert and produce effects that are often underrated and associated with clinical complications in 1.5 to 3 per cent of patients. Some accidents are due to faulty techniques and therefore independent of the contrast medium. The intramural injection is fairly common and is dangerous as it is responsible for nearly one per cent of the serious sequelae (PERRET). An examination performed with a perfectly correct technique may however also produce complications especially if the vascular condition of the patient is precarious. Severe cerebral arteriosclerosis, arterial occlusion with or without hemodiversion or vascular spasm following the rupture of an aneurysm may accentuate the risks.

Apart from any vascular weakness or faulty technique, a certain number of complications directly connected with the contrast medium remain. BROUHA & OLISOV stated twenty five years ago that a contrast medium may alter the cerebral vascular physiology by injuring the blood brain barrier i.e. by increasing the permeability of the cerebral capillaries. This produces cerebral oedema and aggravation of the neurologic signs. On the other hand labelled contrast media have recently proved that if the capillary permeability is increased say by infarcts or tumours some may enter the cerebral parenchyma. The contrast



media possess marked neurotoxicity and their accidental intrathecal injection may cause death. They also possess a marked epileptogenous action which is responsible for convulsions in 0.6 per cent of cerebral angiographies.

Numerous authors have reported secondary cardio-circulatory disturbances after the intracarotid or intravertebral injection of a contrast medium i.e. changes in the heart rate with paroxysmal tachycardia or more frequently bradycardia with secondary hypotension leading to cardiac arrest. The sequelae were particularly frequent following the injection of the contrast media previously used such as diiodinated or the first triiodinated (acetrizoate) products. Cardiac arrest lasting more than ten seconds has been reported and the origin of such a reflex is still being discussed. It is probable that it does not originate at the carotid sinus level but rather in the mesencephalon, anaesthesia of the carotid sinus fails to make it disappear. Premedication in human subjects with atropine of the carotid sinus or section of the vagus nerve in animals considerably reduces the effects. These cardio-vascular reactions appear to be proportional to the neurotoxicity of the contrast medium: their frequency has gradually been reduced by the introduction of substances that are better tolerated. On the other hand the incidence of damage to the central nervous or cardio-vascular system is undeniable in patients suffering from increased intracranial pressure.

Other toxicity factors may also be responsible for complications, for instance the introduction of contrast media may alter the blood biochemically. LITT et coll. produced evidence of alterations in the albumin as well as alpha and beta globulins and an increase in the gamma globulins: clumps of red cells formed when the blood was mixed with the contrast medium: the clump in animals being capable of obstructing small vessels. These authors also stressed that the introduction of the new media had considerably reduced the relative importance of the factors mentioned.

Contrast media have several special features among which may be mentioned the chemical structure of the acid supporting the atoms of iodine, the nature of the salt or salts forming the commercial products and the concentration of iodine and viscosity of the solution expressed in centipoise at 37° C. The media usually employed consist of aqueous solutions of salts of iodinated acid (anions) and a chemical base (cations). As soon as the salts are injected into the blood stream they break up into an acid and a base, each being partly responsible for the global toxicity. The acids entering into the mixtures are all associated with acetrizic acid introduced by WALLINGFORD (1950) (Table I) and modified by their respective inventors. The acids are linked with bases consisting of sodium or calcium ions or organic molecules (methylglucamine or monoethanolamine): calcium has been associated with metrizic acid alone and monoethanolamine with ioxithalamic acid.

Table 1

*Contrast media and their chemical formulae*

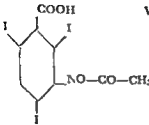
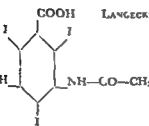
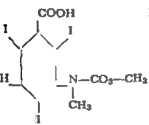
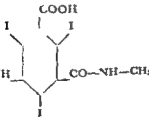
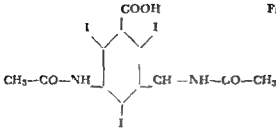
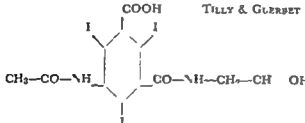
Formulae	Acids
	WALLINGFORD (1950) · Acetrizic Acetamido side chain One NH—CO—CH <sub>3</sub>
	LANGECKER & LARSEN (1954) · Diatrizic Acetamido side chain Two NH—CO—CH <sub>3</sub>
	HOLTERMANN (1961) · Metrizic Hydrogen atom has been replaced by a methyl radical in diatrizic acid
	HOFF (1962) · Iothalam One Methyl carbamyl side chain

Table (cont.)

Formulae	Acids
	<b>FELDER (1955)</b> · Iodamide One CH radical joins the acetamido side chain to the iodized ring
	<b>TILLY &amp; GLERSET (1968)</b> · Iovithalamic One OH radical has been introduced into the molecule

### Experimental techniques

Some of the recent experimental techniques are concerned with the direct action of contrast media on the capillaries of the central nervous system. The best known is the Trypan blue dye test by which BROWN & OLSSON first stressed the dangers of cerebral angiography. Research initiated by GOLDMANN & SPATZ established that normal cerebral capillaries are impermeable to acid dyes. This means that the injection of Trypan blue colours the whole of the body with the exception of the central nervous system. If as a result of localized vascular pathology the capillary permeability is increased in a certain region of the central nervous system, the dye enters the parenchyma of this region to produce a blue colouration.

It is of course well known by those who have investigated the problem of the blood brain barrier that the techniques with vital dyes is effective only in detecting severe lesions. More recent techniques thus employ substances with smaller molecules especially radioisotopes. JEPPESSON does not consider that isotopes are superior to Trypan blue for the investigation of the tolerance of cerebral capillaries to contrast media. The contrary has however been established by several experimenters, the present authors have found that this dye fails to reveal capillary permeability changes when the radioisotopes, and particularly

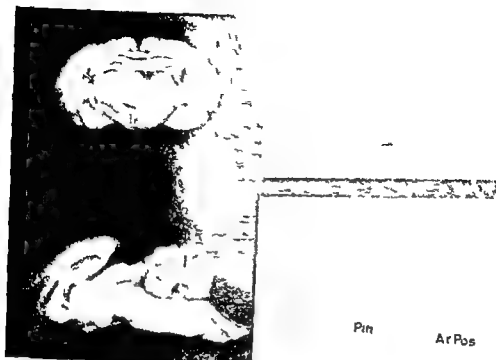


Fig 1 Autoradiograms of cross sections of a normal guinea pig brain after injection of 50 mCi  $^{32}\text{P}$ . This penetrates only the choroid plexuses and hypothalamus (frontal view) and pineal gland and area postrema (lateral view) (Exposure time 17 hours)

$^{32}\text{P}$ , indicate lesions of the blood brain barrier. The passage of radioactive phosphorus appears therefore the more sensitive method of evaluating the importance and size of lesions affecting cerebral vascular permeability (Figs 1-2).

The maximum iodine concentration of the solutions of contrast media that failed to lead to a passage of the  $^{125}\text{I}$  into cerebral parenchyma was therefore determined in guinea pigs following carotid injection in order to indicate their respective toxicity, i.e. that which would affect the blood brain barrier.

The toxicity of contrast media on capillaries and the cerebral parenchyma may also be examined by histologic investigations of the central nervous system. In this connection electron microscopy is much more precise than optical microscopy. The maximum iodine concentration producing no reaction apparent at electron microscopy (in other words cellular toxicity on the central nervous system) was determined.

It was observed that reactions in animals following the carotid injection of contrast media ranged from a simple increase in the endothelial pinocytotic vesicles to swelling of the pericapillary astrocytes leading to their bursting and the



Fig 2 Three autoradiograms of cross sections of guinea pigs brain after intracarotid injection of various contrast media. This technique indicates the importance and the topography of lesions of the blood brain barrier. Cortical (a), hemispheric (b) and whole brain (c) lesions.

formation of artificial perivascular spaces (Figs 3, 4, 5). It is important to stress in this connection that the new commercially available contrast media always cause cerebral oedema which in turn, may produce clinical complications.

The epileptogenic action of contrast media has been investigated by LUNDVOLD who recorded the EEG after their intracarotid injection while HILAL determined the maximum doses leading to clinical convulsions. The action is thus subordinated to the passage of the contrast medium through injury to the blood brain barrier. It is difficult under such conditions to dissociate the relative importance of the two elements. A strongly epileptogenous product but only slightly toxic to the barrier and thus confined to the vascular bed, might for instance give rise to less convulsive phenomena than one less irritant but quickly causing an increase in the capillary permeability and thus an opening to the cerebral parenchyma. This will explain why an investigation of the epileptogenic action of the medium after its pericerebral injection was preferred by the authors. The clinical behaviour of the animals was observed and contrasted with the alterations in the EEG: the latter is of course the more reliable and practical method.

## Results

Experimental investigations have suggested that the secondary effects and neurotoxicity of contrast media are connected. The results obtained with the two first experimental techniques (observation of the passage of P and histology by means of the electron microscope) are summarized in Table 2.

The maximum concentration of solutions causing no cellular lesions revealed by electron microscopy was surprisingly always considerably lower than that damaging the blood brain barrier. The modern ultramicrocopy techniques thus appear to be more valuable than the classic dye test of capillary permeability: they should be applied to all the new contrast media that may be used in cerebral



Fig 3

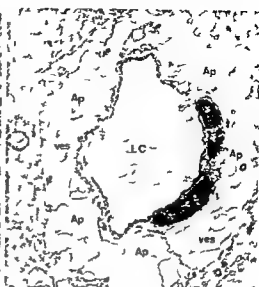


Fig 4

Fig 3 Morphologic peculiarities of capillaries of the brain at electron microscopy. The capillary wall consists of continuous endothelial layer (End). A basement membrane (mb) underlies the endothelial sheet and astrocytic feet (Ap) cover much of the capillary surface. When astrocytic feet do not exist on the capillary surface the component of the neuropil (NP) (nerve cells, oligodendrocytes, myelin sheaths) may be present.

Fig 4 Intracarotid injection of toxic contrast medium. Enlargement of the astrocytic feet with intracytoplasmic vesicles corresponding to the first stage of cerebral oedema (single + in classification).



Fig 5 Severe oedema. Plasma membranes of the astrocytic feet rupture with creation of a pseudoeextracellular space. Flattening of the capillary walls caused by the swollen astrocytes (single ++ in classification).

Table 2

*Results obtained with two experimental techniques in guinea pigs after intracarotid injection of 1 ml of contrast medium*

Acid	Maximum percentage content of iodine not producing			
	Changes in blood brain barrier *P		Anatomical lesion at electron microscopy	
	Na	Mgl	Na	Mgl
Acetrizic (1950)	15	20	12	15
Diatrizic (1954)	23	28	19	24
Metrizic (1961)	24	32	20	28
Iothalamic (1962)	26	36	22	30
Iodamide (1965)	30	32	24	26
Ioxuthalamic (1968)	34	38	27	36

angiography. The determination of the maximum rate of concentration of iodine indicates the level above which oedema in animals and thus complications in human subjects with vascular weaknesses may appear. The neurotoxicity of the various acids varies widely and in toto every new synthesis marks improvement. The most marked changes occurred about fifteen years ago at the time of the synthesis of diatrizic acid and again quite recently during the development of ioxuthalamic acid, each synthesis indicating lowering of about 70 per cent toxicity caused by the acids.

These experiments also reveal the considerable difference that exists as regards toxicity of the salts of sodium and methylglucamine. This toxicity has been reported by many authors and seems to be connected with the biologic behaviour of the ion that gives to these solutions their particular properties of ionization and conductivity. RIVIER has described the counteraction that develops between sodium and calcium. KUTT *et coll.* have reported that a rapid injection of a sodium contrast medium may produce reduction in the blood calcium of more than 25 per cent and lead to a state of tetany in animals. This depression in blood calcium also causes biochemical alterations in the endothelial cellular membrane and changes in the permeability.

Secondary cardiovascular reactions are more frequent and more serious following the intracarotid injection of sodium salts. It seems therefore that these should be avoided in cerebral angiography. SALVESEN & HOLTERMAN, taking advantage of the antagonism of sodium and calcium introduced small amounts of a metrizic salt of calcium into their media. Investigations into the LD<sub>50</sub> by

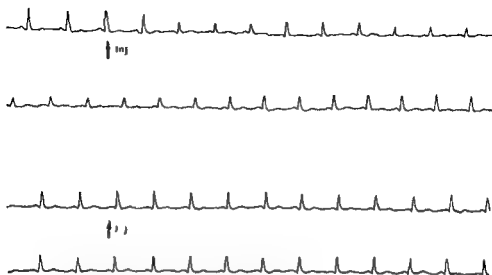


Fig 6 ECG in a patient during intracarotid injection of 10 ml metrizoate sodium ( $\text{Na } 30\%$ ) (above) Flattening of the QRS complex. When calcium is added ( $\text{Na} + \text{Ca } 30\%$ ) the flattening is less marked the protection is effective (below)

intravenous injection indicated that the optimum calcium sodium ratio of the solutions was from six to even times superior to that of the plasma. The protection effect was confirmed in the present material by the  $^{32}\text{P}$  test, electron microscopy and cardiovascular reactions (Fig 6). The fact that it is afforded by the sodium salts of all the iodinated acids but is less apparent with the methylglucamine salts indicates that it rests effectively upon antagonistic physiologic action towards the sodium ions and is independent of the nature of the acid. Even a protected sodium medium is perhaps best avoided in cerebral angiography.

The salts of sodium are associated with the problem of viscosity. Methylglucamine salts produce well tolerated solutions containing a high concentration of iodine although of considerable viscosity. The latter makes injection difficult with small calibre catheters and needles. Clinical investigations have indicated that for the small percentage of iodine contrast media of low viscosity produce in the presence of circulatory obstruction relatively more complete vascular filling than more viscous solutions. The optimum viscosity should be as near as possible to that of the blood, taking the latter as 4 cP at  $37^\circ\text{C}$ . a concentration of iodine of 26 to 28 per cent for the sodium salts and of only 26 to 28 per cent for the methylglucamine salts may be employed. To obtain greater concentrations while retaining the same viscosity the sodium and methylglucamine salts must be



Table 3

*Clinical manifestations after pericerebral injection (0.03 ml) of various contrast media in the guinea pig*

Acid 280 mg 1 ml methylglucamine salt	Number of animals in a batch	Agitation	Jacksonian fits	Status epilepticus	Death
Diatrizoic	3	5	5	5	5
Metrizoic	3	3	5	5	0
Iodamide	3	5	5	5	1
Iothalamic	3	3	3	2	0
Ioxithalamic	3	5	0	0	0

Table 4

*Alteration in the FEG after pericerebral injection (0.035 ml) in the guinea pig*

Chemical names	Salts	Trade name	Iodine concentration	Number of crisis EEG
Iothalamate	Mgl	Contrix 38	28	5
Ioxithalamate	Mgl + M Ethan	Vasobrix 32	28	0
Ioxithalamate	Mgl + M Ethan	Vasobrix 32	32	1

mixed and the aspect of tolerance neglected. For instance, two solutions of diatrizoic acid have a concentration of about 4 cP. The first (Urografin 60) contains Na Mgl in a 1:6 ratio with an iodine strength of 29 per cent. The central nervous system tolerance to this is reasonable. The second solution (Urovison) is also prepared from sodium and methylglucamine salts but in an inverse ratio i.e. 6:1 which enables an iodine concentration of 32 per cent to be reached while retaining a viscosity of 4 cP. Unfortunately this solution is toxic in a high degree to the central system and is unsuitable for cerebral angiography.

A compromise between the concentration of iodine and the viscosity appears to have been effected by TILLY & CUERBET with ioxithalamic acid and the monoithalolamine salt. The product has a viscosity equal to that of the sodium agents and possesses a tolerance ranking between those of the sodium and methylglucamine salts. The introduction of a new salt combined with the exceptional tolerance of ioxithalamic acid suggested the synthesis of a new product and one containing as much as 32 per cent of iodine, a viscosity of 4 cP.

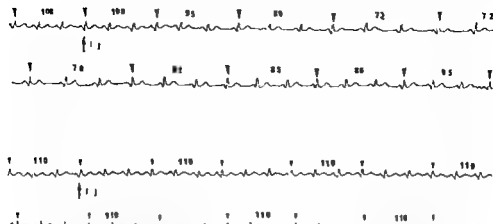


Fig 7 ECG during intracarotid injection of 10 ml of metrizoate sodium 37%. Immediate marked bradycardia from 108 to 72/min heart rate (above). After intracarotid injection of iohalamate monoethanolamine and methylglucamine 37% no modification in the heart rate occurs (below). This product with a new salt seems to cause no appreciable effect on the cardiovascular system.

and well tolerated by the central nervous system. Experimental investigations in animals indicated that it was even possible to increase the iodine concentration up to 34 or 36 per cent without the appearance of signs of neurotoxicity.

The epileptogenic action of contrast media is well known. The experiments of KODAMA & CAMPBELL established that substantial differences between the various acids also existed in this field: the sodium salts are evidently more epileptogenic than those of methylglucamine. The effects of pericerebral injections of 0.03 ml methylglucamine salts of triiodinated acids, each acid investigated in several batches, are presented in Table 3.

The EEG was recorded for the best tolerated acids (Table 4). The investigations confirmed the reduced epileptogenic action of the methylglucamine salt of iohalamate (Contrix 28) and indicated also that if the salts of iohalamate (Vasobrix) are not without such action, it is reduced for a higher concentration of iodine.

This recent contrast medium derivative of iohalamic acid (Vasobrix 32) containing a new salt (monoethanolamine) was investigated without premedication from the viewpoint of cardiovascular reactions following cerebral angiography.

In 60 carotid angiographies no modification of the ECG occurred in about half the cases (Fig 7). Slight tachycardia occurred in 26 cases, in only 2.7 per cent of which did the total alteration in rhythm exceed 20 per cent; this was

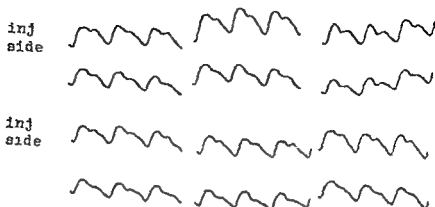


Fig 8 Rheoencephalography during intracarotid injection of Ringer's solution (below) or of mannitol 20% (above). From left to right: Before, one and two minutes after injection, respectively. The Ringer solution causes no modification of the REC, but the hypertonic solution of mannitol 20% produces an increase in the rheoencephalographic curve on the side of injection lasting one minute.

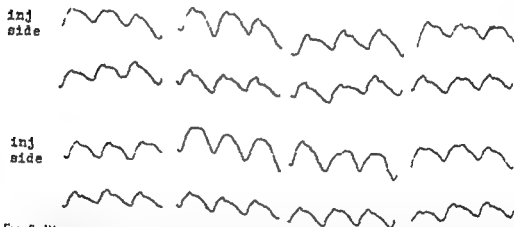


Fig 9 Rheoencephalography during intracarotid injection of contrast media. The diatrizoate sodium methylglucamine (Urografin 60) (above) causes the same modifications as mannitol 20%. From left to right: Before, one, two and three minutes after injection, respectively. The ioxothalamate monoethanolamine/methylglucamine (Vasbrix 32) (below) produces an unilateral increase in the rheoencephalographic curve lasting more than two minutes.

probably due to inadequate anesthesia. Bradycardia was recorded in 8 cases only, in none of which was the heart rate altered by more than 20 per cent. Vertebral angiography in 23 of 40 cases, i.e. more than half the cases, produced no modification in rhythm. 14 presented moderate tachycardia, and only 3 cases bradycardia under 10 per cent of the heart rate, respectively.

The new salt seems to cause no effect upon the cardiovascular system.

A final aspect of the contrast media is their vascular physiology is their vasomotor action. Animals were investigated to observe modifications in the cerebral vessels (Brown, 1934) by measuring certain parameters such as blood pressure, heart rate and blood flow (HILAL, LINDGREN, et coll.). It is generally admitted that the intravascular injection of a contrast medium alters the cerebral circulation by increasing the flow. HILAL insisted on the importance of the hypertonicity of the solutions in these phenomena. As to the vasomotor action, if the mono- and triiodinated contrast media appear to have a vasoconstrictor effect, the triiodinated media should produce vasodilatory changes especially upon branches of the external and internal carotid arteries. This theory has been confirmed by HUBER who examined the smaller cerebral arteries and by GREITZ who dealt with the veins.

The present author investigated the modifications in the cerebral plethysmogram (rheoencephalography) recorded in human subjects continuously during and after the intracarotid injection of various contrast media. As in animal experiments, the intracarotid injection of a physiologic solution does not alter the cerebral plethysmogram although hypertonic solutions produce an increased blood flow in the affected hemisphere (Fig. 8). All the contrast media with an equal iodine concentration and equal viscosity act in increasing the rheoencephalographic curve: the product compounded from iothalamic acid (Vasobrix 32) causes an increase lasting up to four or five minutes (Fig. 9). It is difficult to state whether these modifications arise from cerebral vasodilatation or by an increase in the blood flow, but they appear to be comparable to those observed after the intravascular injection of other drugs with a vasodilatory action.

Several points remain to be elucidated, particularly the roles of the chemical natures of the acid or salt and certain physical characteristics of the solutions. It is known, for instance, that chlorhydrate of methylglucamine (when not used as a contrast medium) or hypertonic solutions have a vasomotor action although it is probable that this may differ under pathologic conditions. RAYBOR has reported that even if derivatives of diatrizoic acid do not provoke pain, they maintain for some time any contraction present before their injection.

Three reported materials are compared in Table 5 with a view to determining whether the experimental improvement has been confirmed in the clinical employment of modern contrast media. The figures indicate the importance of the base since when the solutions of sodium salt of the diatrizoic acid (according to PERRET) give way to those of the methylglucamine salts of the same acid (according to TORMA), the complications decrease. The importance of the nature of the acid itself is indicated by comparing the diatrizoic acid salts of TORMA

Table 5

*Clinical complications in cerebral angiography*

Authors	PERRET (1966) 2 591 cases	TORMA (1968) 1 000 cases	GONSETTE et coll 2 000 cases
Acids	Diatrizoic	Diatrizoic	Iovithalamic
Salts	Na	Mgl/Na 6/1	Mono ethan /Mgl 2/1
Iodine content	27 °	29 °	32
Transitory neural defic.	96 cases (1.8 %)	8 cases (0.8 %)	0
Permanent neural defic.	39 cases (0.8 %)	11	0
Convulsions	33 cases (0.6 %)	5 cases (0.5 %)	2 cases (0.1 %)

with those from 2 000 angiographies performed with a recent contrast medium obtained from iovithalamic acid

Important progress has thus been made during the last fifteen years in reducing the toxicity upon cerebral tissue produced by capillary permeability and bioelectric influences

### Conclusion

The contrast media employed in angiography appear to have toxic effects not only on the physiology of the cerebral vessels but on the central nervous system itself. The influences have not always been considered in the synthesis of the contrast media designed for cerebral angiography, tests of general toxicity do not necessarily convey an idea of the possible local effects. It is essential that the pharmaceutical industry should appreciate these factors and develop better tolerated products. Notable progress in this direction has been made with a recent contrast medium. Improvement is still to be expected but whether it will eventually lead to the production of biologically inert products of a form it is premature to forecast. It is unlikely that there will be polymerization of the acids already widely known such as those investigated by HILAL and BJORK. The reduction in the hypertonicity of the solutions is interesting but always attended with a notable increase in their viscosity. There will be rather non ionic water soluble contrast media (ALMEX) which may offer the advantages of low viscosity, low tonicity and improved biologic inertness.

### SUMMARY

The tolerance of the central nervous system to various contrast media has been investigated with special action on the blood brain barrier, electron microscopy (cellular toxicity) and EEG alterations (after pencerebral injection). The media may increase the permeability

of the cerebral capillaries and provoke cerebral oedema thus may explain many of the usual clinical complications. The failings of the usually employed in cerebral angiography are considered and a new and improved contrast agent is presented.

### ZUSAMMENFASSUNG

Die Toleranz des Zentralnervensystems gegenüber verschiedenen Kontrastmitteln wurde mit  $^{51}\text{Cr}$  (Wirkung auf die Blut-Gehirn-Schranke Elektronenmikroskopie, zelluläre Toxizität) und EEG Veränderungen (nach perivaskulärer Injektion) untersucht. Die Mittel können die Permeabilität der Kapillaren des Gehirns erhöhen und ein Gehirnoedem hervorrufen. Dieses kann viele der gewöhnlichen klinischen Komplikationen erklären. Die Nachteile der Mittel, die bei der zerebralen Angiographie gewöhnlich verwendet werden, werden betrachtet und ein neues und verbessertes Präparat vorgeschlagt.

### RÉSUMÉ

L'auteur a étudié la tolérance du système nerveux central à différents moyens de contraste en utilisant le  $^{51}\text{Cr}$  (action sur la barrière hémato-encéphalique, la microscopie électronique (toxicité cellulaire) et les alterations EEG (après injection pericébrale). Ces moyens de contraste peuvent augmenter la perméabilité des capillaires cérébraux et dans des conditions pathologiques provoquer un œdème cérébral. L'auteur étudie les inconvénients des moyens de contraste employés habituellement pour l'angiographie cérébrale et présente un produit nouveau et amélioré.

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AUS DEM ZENTRAL RÖNTGENINSTITUT DES KATHARINENHOSPITALS DER STADT  
STUTT GART (DIREKTOR PROF. F. HEUCK) DEM INSTITUT FÜR ANGEWANDTE  
PHYSIK DER UNIVERSITÄT KIEL (DIREKTOR PROF. W. KROEBEL) UND DEM INSTITUT  
FÜR BIOMEDIZINISCHE TECHNIK AN DER UNIVERSITÄT STUTT GART DEUTSCHLAND

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## DIE RÖNTGEN DENSITOMETRIE DER HIRNSTROMBAHN

VON

F. HEUCK, U. PIEPGRAS und K. VANSELOW

In letzter Zeit wurden verschiedenartige radiologische Untersuchungsverfahren entwickelt um semiquantitative oder quantitative Messungen der Kreislaufzeit des Blutstromvolumens und der Stromungsphänomene im arteriellen oder venösen Schenkel der Strombahn des großen Kreislaufes durchführen zu können. Das Studium der Blutverteilung auf die Parallelkreislaufe und damit die Versorgung lebenswichtiger Organe (Hirn, Niere, Leber) am nicht narkotisierten wachen Menschen ist von besonderer Bedeutung für die klinische Medizin.

Die bisher bekannten Methoden der radiologischen Kreislaufanalyse basieren auf dem Prinzip die Fortbewegung entweder eines Kontrast Bolus im Gefäßlumen mit Hilfe von Serienröntgenaufnahmen und Röntgenkinofilmen hoher Bildfrequenz festzuhalten oder eines Isotopen Bolus also eines radioaktiv markierten Stoffes durch die Strombahn mit Meßgeräten zu verfolgen.

Das höchste Auflösungsvermögen besitzen solche Meßverfahren die das Röntgenbild, das Röntgenkinobild oder ein Bildverstärkerfernsehbild der Auswertung zugrunde legen. Die im Röntgenbild oder im Bildbandspeicher enthaltene Information kann mit Hilfe der Photometrie oder Densitometrie ana-



lysiert werden. Bei Verwendung der Röntgenkinematographie sprechen wir von Cine Densitometrie, bei Verwendung der Bildverstärkerfernsehtechnik von Video Densitometrie. Beide Methoden sind zur Kreislaufanalyse herangezogen worden. Neben einem großen apparativen Aufwand und hohen Kosten besitzt die Video Densitometrie gewisse Nachteile, auf die hier nicht näher eingegangen werden kann (HEINTZEN et coll 1967, RUTISHAUER et coll 1967, HEUCK & VANSELOW 1970).

**Methodik.** Die Methoden einer direkten Densitometrie müssen zahlreiche Schwierigkeiten überwinden (VANSELOW et coll 1968, HEUCK & VANSELOW 1970). Mit der Elimination von Störfaktoren bei direkten Meßverfahren der Cine Densitometrie oder Video Densitometrie haben sich WOOD et coll (1960/64), HEINTZEN et coll (1967), HILAL (1966) und RUTISHAUER et coll (1967) beschäftigt; ohne daß es bisher gelungen wäre, wesentliche Verbesserungen in der Meßtechnik zu erreichen. Die Untersuchungen wurden im Tierexperiment und am Menschen vorgenommen. Zur Beseitigung der Störfaktoren sind außerordentlich kostspielige komplizierte apparative Einrichtungen erforderlich, so daß eine praktische Verwendung in der klinischen Routinediagnostik kaum möglich erscheint.

Es war daher ein vordringliches Anliegen, ein Meßverfahren zu entwickeln, mit dessen Hilfe die obengenannten Probleme der direkten Densitometrie eliminiert werden konnten. In unserem Arbeitskreis wurde eine Methode zur Analyse der im Röntgenbild gespeicherten zahlreichen Informationen erarbeitet, der ein nicht unbekanntes Prinzip der physikalischen Meßtechnik zugrunde liegt. Das Verfahren basiert auf der Quotientenbildung der Meßwerte zweier räumlich getrennt liegender Meßpunkte desselben Bildes (VANSELOW et coll 1968, GUILINO & MÜLLER 1970). Als Meßzellen dienen Photowiderstände, die paarweise angeordnet sind, so daß die jeweils zwischen zwei Punkten bestehenden Helligkeitsdifferenzen in einen Photostrom unterschiedlicher Spannung umgewandelt werden. Als wesentliches Bauelement ist eine Brückenschaltung vorhanden, die einerseits zwei konstante Widerstände, andererseits die beiden variablen Photowiderstände für die Messung selbst enthält. Es kommt nur dann zu einer Spannungsänderung, wenn sich auch die Photowiderstände in unterschiedlichem Ausmaß ändern. Bei gleichsinniger Änderung des Widerstandswertes der paarweise angeordneten Photowiderstände um denselben Faktor tritt eine Spannungsschwankung in der Brückenschaltung nicht auf. Es können hiermit Störungen und Untergrundschwankungen ausgeschaltet werden. Alle nicht durch die Kontrastsubstanz hervorgerufenen Helligkeitsänderungen werden eliminiert, so daß ausschließlich das anflutende Kontrastblut und die Kontrastkonzentration erfaßt werden. Jede auftretende Spannungsänderung wird über einen Mehrfachsreiber in eine Kurve transformiert. Dieses Meßprinzip ist allen in hier

bekannten direkten Meßmethoden; insbesondere der absoluten Photometrie und der absoluten Video-Densitometrie überlegen

Die Auswertung der Röntgenkinofilme (Aufnahmefrequenz 48 Bilder/Secunde) erfolgte mit dem Ziel aus der gemessenen Stromungsgeschwindigkeit also den Kreislaufzeiten der gesamten Hirnstrombahn oder einzelner Abschnitte derselben eine Stromungskarte zu gewinnen. Dabei wurde versucht die Frontgeschwindigkeit, die Gruppengeschwindigkeit und die Phasengeschwindigkeit der Blutstromung zu berücksichtigen. Ferner wurde das Blutstromvolumen in der A. carotis interna aus der gemessenen Stromungsgeschwindigkeit und dem im Serienangiogramm bestimmten Gefäßquerschnitt errechnet. Die Messungen des Gefäßlumens der A. carotis interna und ihrer Verzweigungen muß den Vergrößerungsfaktor berücksichtigen, der durch den gegebenen Fokus-Objekt- und Objekt-Film-Abstand resultiert. Unter Beachtung dieser Fehlermöglichkeiten ergaben die Messungen in 32 von bisher 38 Fällen einen Lumen-Durchmesser von 4—5 mm im Mittel also 4,5 mm. Als Kontrastsubstanzen für die Angiographie wurden Urografin 60 %ig (Schering Berlin) und Conray 60 %ig (Byk-Culden Konstanz) verwendet, die in den letzten 5 Jahren bei über 2400 Carotisangiographien keinerlei Komplikationen verursacht haben. Die pharmakologischen Nebenwirkungen der verwendeten Kontrastsubstanzen auf die Hirnstrombahn sind von verschiedenen Arbeitsgruppen untersucht worden, die zu sehr unterschiedlichen Ergebnissen gelangten (GREITZ 1956/68, GREITZ & SYK 1968, HILAL 1966, HUBER 1967, TINDALL et coll. 1965, ZINGESSER et coll. 1968). Es wäre möglich, daß diese Differenzen durch die verschiedenen Methoden bedingt sind. Bleiben das methodische Vorgehen, die injizierte Kontrastmitteldosis und die Vorbereitung des Patienten sowohl bei Untersuchungen an Gesunden wie an Kranken gleich, so kann ein eventuell auftretender geringer Fehler vernachlässigt werden. Wichtig erscheint es, die Untersuchungen möglichst am nicht narkotisierten also wachen Menschen vorzunehmen. Nach Modelluntersuchungen von GIDLUND (1956), KLINGLER (1959) soll eine Druckänderung, die während der Injektion des Kontrastmittels in das Gefäß auftritt, belanglos sein. Die Zunahme des Druckes während der Carotisangiographie ist besonders dann gering, wenn eine kleine Kontrastmittelmenge von 5—10 ml verwendet wird, so daß sie praktisch vernachlässigt werden kann.

Auf Einzelheiten der Aufnahme-Apparatur und Aufnahme-Technik, der Auswertung des Filmmaterials und der Densitometer-Kurven kann an dieser Stelle nicht eingegangen werden, entsprechende Angaben sind in bisher erschienenen Mitteilungen zu finden (HELCK et coll. 1969, HELCK & VANSELON 1970, PIEPGRAS et coll. 1968). Die beschriebene Methode der Cine-Densitometrie erlaubt die Bestimmung verschiedener Kreislaufgrößen in jedem Abschnitt der Strombahn und die Analyse der Geweb-durchblutung in ihr.

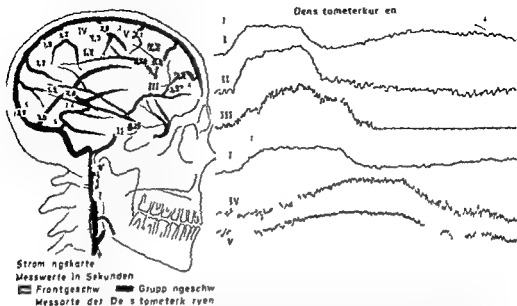


Abb. 1. Schematische Darstellung der Blutströmungsgeschwindigkeit im Hirnkreislauf nach den Ergebnissen der Densitometrie in einer Strömungskarte (links). Daneben sind die Densitometerkurven von 5 Meßorten über dem Hirnschädel dargestellt: I und II über der A. carotis interna (Blutstromvolumen 40 ml/s); III über der A. cerebri anterior; IV und V Gewebsdensitometrie (40-jähriger Mann).

kleinen Arealen. Das zeitliche und räumliche Auflösungsvermögen der Röntgen-Densitometrie ist in den makroskopischen Dimensionen des Blutkreislaufes annähernd unbegrenzt, da der kleinste Durchmesser der Meßzellen 1 mm beträgt und bei entsprechend hoher Lichtstärke des Filmprojektors eine beliebige Vergrößerung des Röntgenkinobildes vorgenommen werden kann.

Die vorgelegten ersten Befunde der Densitometrie des Hirnkreislaufes von Gesunden und Kranken wurden an 38 Röntgenkinofilmen (48 Aufnahmen/Sekunde) erarbeitet. Die Filme wurden jeweils vor Durchführung der diagnostischen Hirnangiographie (AOI-Serie) angefertigt. Die Meßstrecken im Bereich der A. carotis interna und ihrer Verzweigungen, die Meßorte über dem Großhirn und die Meßpunkte im venösen Schenkel der Hirnstrombahn sind in den Skizzen dargestellt.

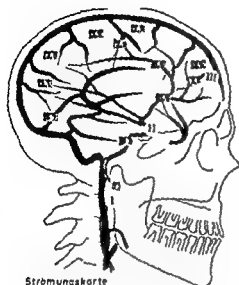
### Befunde

Im Bereich der Hirnstrombahn sind Kreislaufzeiten der verschiedenen Phasen des Organkreislaufes und das Blutstromvolumen in der A. carotis interna bei Gesunden und Kranken untersucht worden. In einer früheren Mitteilung

Tabelle  
Meßergebnisse

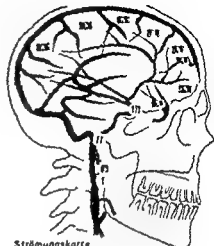
Alter Geschl	Diagnose Befund	Blutströmung Art. carot. int.		Kreislaufzeiten des Gehirns		
		ml/s	cm	A. Gew.	A. Venen	A. Sinus
Gesunde						
49 ♀	o.B.	51	76			
47 ♂	o.B.	57	29	0.64—1.7	2.6—3.7	
40 ♂	o.P.	62	21	0.67—1.1		
34 ♀	o.B.	57	37	0.9—1.6	2.0—2.9	7.7—3.4
46 ♂	o.B.	40	23	1.16	2.8	
43 ♀	o.B.	42	24	1.0	2.4	
Gefäßerkrankungen						
58 ♀	Media Verschluss	26	75	2.0—7.5	2.8	4.3—5.1
61 ♀	Media Verschluss	25	31	2.3		4.5
56 ♂	Stenose A. car. int.	21	16	2.8		
53 ♂	Arteriosklerose	0.64	4.3	1.0	3.5—4.0	
54 ♀	Arteriosklerose	1.3	9.7	7.3	4.1—4.3	5.7—7.1
17 ♀	Cerebr. Anfall	0.9	8.5	2.6	2.9—3.7	3.7—4.6
25 ♂	Cerebr. Anfall	4.8	30	0.7—1.1	1.3—3.0	3.3—5
Raumfordernde Prozesse						
20 ♀	Balkenlipom	45	35	1.6	2.7—2.9	2.4—3.8
36 ♀	Verdacht auf Tumor	4.3	24	1.1	1.7	
64 ♂	Tumor oder Metast.	3.5	20	2.2	2.7	
43 ♀	Meningeom rechts	2.0	20	2.5	4.8	4.3
37 ♀	Verdacht auf Tumor	3.2—4.0	26—33	1.3—7.4	3.6—4.3	3.5—5.3
58 ♀	Glioblastom links	7.9	46	1.7	3.0	
30 ♀	Astrocytom (Op.)	4.2	26	0.8—1.4	2.9—3.7	7.9—3.7

(HEUCK & PIEPGRAS 1967) wurde auf die Information hingewiesen, die eine Analyse der densitometrisch gewonnenen Kontrastmitteldünnungskurven geben kann. Es ist dabei insbesondere die Gewebedurchblutung (Parenchymphase) studiert worden, doch sind bisher quantitative Messungen mit Hilfe eines geeichten Referenzsystems an einem größeren Krankengut nicht durchgeführt worden. Derartige Messungen sind nur mit dem Einsatz von Computern sinnvoll und wurden im Institut für Biomedizinische Technik begonnen. Allein aus dem Kurvenverlauf der Gewebs-Densitometrie können jedoch jetzt schon



Strömungskarte

■ Messwerte in Sekunden bei Gruppengeschw.  
Messorte der Densitometerkurven



Strömungskarte

■ Messwerte in Sekunden bei Frontgeschw.  
Messorte der Densitometerkurven

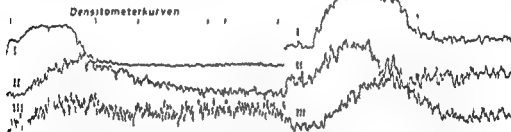


Abb. 2 Strömungskarte und Densitometerkurven bei einer Gefäßkrankung mit Media Ver-  
schluß (59-jähriger Mann). Die Verlangsamung  
der Blutströmung ist deutlich festzustellen I  
und II über der A. carotis interna (Blutstrom-  
volumen 26 ml/s) III Isophor

Abb. 3 Veränderungen der Strömungs-  
geschwindigkeit und des Blutstromvolumens  
bei pyramitärem Meningeom (46-jähriger  
Mann) I II und III über der A. carotis  
interna (Blutstromvolumen 20 ml/s)

Hinweise auf eine gestörte Zirkulation entnommen werden (Pieperas et coll  
1968).

Die in verschiedenen Abschnitten der Hirnstrombahn densitometrisch ermit-  
telten Strömungsgeschwindigkeiten geben Hinweise auf die Hamodynamik des  
Organkreislaufes. Es ist möglich eine Strömungskarte nach topographischen  
Gesichtspunkten anzufertigen ohne die anatomischen Varianten des arteriellen  
und venösen Schenkels der Strombahn im einzelnen berücksichtigen zu müssen.  
Die Densitometrie erfaßt die Ankunft und den Durchtritt des Kontrast Bolus  
am Meßort, legt also die Frontgeschwindigkeit und damit den Zentralstrom im

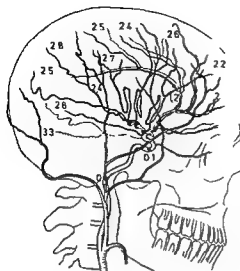
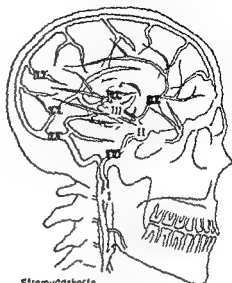


Abb 4 Densitometrisch gewonnene arterielle Stromungskarte bei metastatischem Tumor links frontal (64-jähriger Mann). Abnahme der Strömungsgeschwindigkeit als Ausdruck einer Zirkulationsstörung. Blutstromvolumen der A. carotis interna 35 ml/s

Gefäßrohr zugrunde. Eine Gegenüberstellung der Stromungskarte des Gesunden (Abb 1) und der Stromungskarten bei Hirnkrankungen läßt Veränderungen der Blutstromung erkennen und gibt damit einen Einblick in die Pathophysiologie des Hirnkreislaufes. Bei Gefäßerkrankungen der Hirnstrombahn findet sich meist neben einer Abnahme des Blutstromvolumens in der A. carotis interna eine verlangsamte Blutstromung (Abb 2 Tabelle). Die Stromungskarte gutartiger Tumoren, die langsam gewachsen und zeigt oft nur geringe oder kaum erkennbare Abweichungen, während bösartige Geschwülste oder metastatische Tumoren deutliche Veränderungen der Stromungskarte aufweisen können (Abb 3—5). Meist ist eine Verlangsamung der Blutstromung festzustellen. Besonders betont sei, daß es sich hierbei zunächst nur um eine globale Information zur Hamodynamik eines Organkreislaufes handelt, deren subtile Analyse noch aussteht.

Die Messungen des Blutstromvolumens — also der Durchflußmenge in einem umschriebenen Gefäßabschnitt — wurden nicht nur unter Zugrundelegung der Frontgeschwindigkeit des Kontrast Bolus durchgeführt, sondern es wurden die Gruppengeschwindigkeit und die Phasengeschwindigkeit mit berücksichtigt (Abb 6). Als wahre Transportgeschwindigkeit kommt nur die Gruppengeschwindigkeit in Frage und diese allein bestimmt die Durchflußmenge. Die gemessenen Geschwindigkeiten beziehen sich auf die Ebene der Röntgenfilme, so daß die tatsächliche Geschwindigkeit meist etwas höher anzusetzen ist. Die Frontgeschwindigkeit hängt stark von den örtlichen Gegebenheiten und dem Verlauf



Stromungskarte

■ Messwerte in Sekunden bei Frontgeschw.  
 ■ Messorte der Densitometerkurven

Densitometerkurven

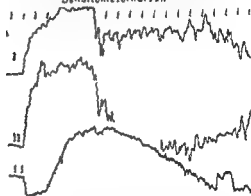


Abb. 3. Bei einem zentral gelegenen Angiom zeigt die Densitometerkurve die sehr rasche Auffüllung des Gefäßknäuels, eine frühzeitige Venenfüllung und die verlangsamte Entleerung der blutgefüllten Räume I und II über der A. carotis interna. III Angiom.

des Gefäßrohres ab. In solchen Gefäßarealen, deren topographisch anatomische Situation überschaubar und in denen die Frontgeschwindigkeit der Blutströmung der Gruppengeschwindigkeit annähernd gleich ist, ist die Auswertung der Röntgenkinofilme einfacher.

Die Klinik interessiert die Blutversorgung des Kopfes, also das Blutstromvolumen der Hirnarterien, ferner die Blutverteilung im Erfolgsorgan, die Durchflußgeschwindigkeit und der venöse Abstrom des Hirnblutes. Einige Meßwerte der gesunden und kranken Hirnstrombahn sind in der Tabelle zusammengeestellt. Besonders eindeutige Veränderungen liegen bei Gefäßerkrankungen vor.

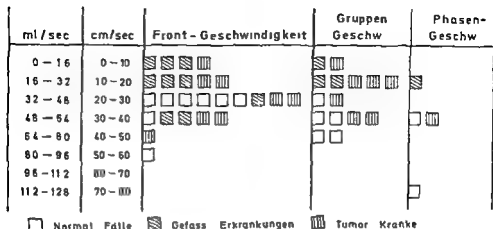
Arteria carotis interna

Abb 6 Zusammenstellung der Strömungsgeschwindigkeit bei Gesunden und Kranken mit den errechneten Werten des Blutstromvolumens in der A. carotis interna

Hirntumoren oder Hirnmetastasen ergeben dagegen Abweichungen sowohl nach oben als auch nach unten. Eine weitergehende Analyse wird erst ein größeres Krankengut erlauben. Die bisher mit einer neuen Methode der Röntgen-Densitometrie erarbeiteten Befunde lassen zunächst die schwierigen Probleme der Hamodynamik außer acht. In weiteren Untersuchungen, die nur mit Hilfe von Computern und unter Mitarbeit von Strömungsingenieuren und Physikern möglich sind, sollen einige der zahlreichen noch unbeantworteten Fragen der Physiologie des Hirnkreislaufes vom Menschen bearbeitet werden.

### Besprechung

Die cerebrale Kreislaufzeit und das Blutstromvolumen in der A. carotis interna lassen sowohl bei Gesunden als auch bei Gefäßerkrankungen und Neubildungen im Großhirn eine gewisse Abhängigkeit voneinander erkennen. Nur wenige Befunde konnten nicht eingeordnet werden. Die Bestimmung der Kreislaufzeiten erfolgte in Anlehnung an GREITZ (1968), der als Hauptzirkulationszeit das Zeitintervall zwischen der Maximalfüllung des Carotidsiphons und der parietalen Venen bezeichnet. Die Zeitspanne umfaßt die arterielle Phase, die kapillare Phase und den Anfangsteil der venösen Phase des Kontrastmitteldurchflusses. Die von GREITZ & SIK (1968) gefundenen Werte der Zirkulationszeit bei Gesunden lagen um 35 Sekunden ( $\pm 0,5$  Sekunden) und stimmen mit den eigenen densitometrischen Meßwerten überein. Es ist be-



merkenswert daß die allein durch Auswertung von Einzelbildern der Serienangiogramme gefundenen Kreislaufzeiten ähnliche Befunde ergeben haben. In vergleichenden Untersuchungen haben GROSSQVIST & GREITZ (1969) unter Anwendung der Isotopenmethoden von INGVAR & LASSEN (1962) zwei Phasen der Blutströmung des Hirnkreislaufes festgestellt. Die Phase der schnellen Strömung (etwa analog der Frontgeschwindigkeit bei densitometrischen Analysen) entspricht wahrscheinlich der Herzsystole, während die Phase der langsamen Strömung (zu vergleichen mit der Gruppengeschwindigkeit der densitometrischen Analyse) vorwiegend in die Diastole fällt.

Die Kreislaufzeiten weichen bei Hirnerkrankungen vor allem bei Gefäßprozessen, Abweichungen auf. Bei Tumoren waren Verlängerungen der Kreislaufzeit festzustellen, besonders deutlich beim gesteigerten Hirndruck. Auch RALSCH & SCHIEFER (1956) fanden bei erhöhtem Hirndruck eine Verlängerung der kapillaren Phase bis zu 4,2 Sekunden und der venösen Phase. Eine verlangsamte Zirkulation und ein Absinken der Hirndurchblutung konnten bei erhöhtem Hirndruck auch mit der Methode von KETI & SCHMIDT (1945) nachgewiesen werden. Die Pathophysiologie einer Verminderung der Hirndurchblutung bei Gefäßerkrankungen ist hinsichtlich der Kollateralkversorgung als Reservefunktion aller noch funktionstüchtigen Strombahnabschnitte sowie der Ausdehnung und Lokalisation der Stenose noch weitgehend unbekannt. Das hohe Auflösungsvermögen der Densitometrie erlaubt eine wesentlich subtilere Analyse als andere Meßverfahren, die an einem größeren Krankengut vorgenommen werden soll.

Das Blutstromvolumen der A. carotis interna betrug beim Gesunden etwa 4—5 ml/Sekunde. Diese Meßwerte stimmen mit Ergebnissen anderer Autoren überein. KLINGLER (1959) fand als häufigste Durchblutungsgröße in der A. carotis communis Werte von 6—8 ml/Sekunde und in der A. carotis interna Werte von 5—6 ml/Sekunde. HUBER (1968) fand ein Blutstromvolumen in der A. carotis interna von etwa 330 ml/Minute, umgerechnet also 5,5 ml/Sekunde. Bei cerebro-vascularen Störungen fand HUBER (1968) ein Absinken des Blutstromvolumens in der A. carotis interna auf 70 % des Normalwertes und bei einem Verschuß der A. cerebri media auf 50 % des Normalwertes. Ältere Menschen mit oder ohne Hypertonie zeigten ein vermindertes Stromvolumen. Bemerkenswert ist die Verminderung des Blutstromvolumens bei cerebralen Anfallsleiden. Die wenigen Meßwerte zeigen eine gute Übereinstimmung mit den Ergebnissen einer globalen Messung der Hirndurchblutung (z. B. nach der Methode von KETI & SCHMIDT 1945). Allerdings erlaubt die kleine Zahl der bisher untersuchten Kranken noch keine verbindlichen Aussagen über pathognomonische Veränderungen der Funktionen des Hirnkreislaufes.

Die regionale Hirnzirkulation ist von einer so großen klinischen Bedeutung daß jede Methode die einen brauchbaren Meßwert liefert geprüft werden sollte. Mit den meisten Iotopen Meßverfahren können nur globale Meßwerte über einem größeren Hirnareal gewonnen werden während die Frage nach örtlichen Differenzen der Gewebsdurchströmung unbeantwortet bleibt. Messungen der lokalen Hirnzirkulation mit Hilfe von 32 oder 33 kleinen Detektoren (12 mm  $\phi$  und 10 mm Bleikollimator) haben SVENSDOTTIR et coll. 1969 durchgeführt. Mit dieser Methode wird ein höheres Auflösungsvermögen erreicht.

Die mit Hilfe der Angio-Densitometrie ermittelten Kreislaufdaten des Gehirns stellen einen wichtigen Indikator für verschiedenartige Störungen dieses Organkreislaufes dar und sind vor allem dann von großem Wert wenn die Serien-Angiographie einen normalen anatomischen Befund aufweist. Eine verfeinerte Meßtechnik wird Untersuchungen in kleineren Gefäßästen und die Bestimmung der Gewebdurchblutung ermöglichen. Die Analyse der Densitometerkurven mit Hilfe von Computern bietet die Möglichkeit ohne besonderen Aufwand an Zeit auch andere Meßwerte wie Blutdruck, Pulskurve, Blutgasanalysen usw. zu den Befunden der Densitometrie in Beziehung zu setzen und gemeinsam auszuwerten. Eine Kombination der Densitometrie mit Iotopenmeßverfahren (Untersuchungen des  $O_2$ -Verbrauches im Gewebe nach TER POGOSSIAN et coll. 1969) erscheint besonders interessant.

In Zukunft wird die morphologische Darstellung der arteriellen und venösen Strombahn durch eine Funktionsanalyse des interessierenden Kreislaufareals ergänzt werden können. Die vorgelegte Methodik wird mit Hilfe der Röntgen-Kinematographie hoher Bildfrequenz auch eine Analyse von Strömungsphänomenen in Arterien und Venen erlauben und völlig neue Einblicke in die Physiologie und Pathophysiologie des Kreislaufes am lebenden, nicht narkotisierten Menschen ermöglichen.

## ZUSAMMENFASSUNG

Das Prinzip einer neuen Methode der Röntgen-Cine-Densitometrie zur Analyse des Hirn-Kreislaufes am Menschen wird dargestellt. Die ersten Untersuchungsergebnisse über die Blutströmungsgeschwindigkeit, das Blutstromvolumen (in der A. carotis interna) und die Kreislaufzeiten des Gehirns werden besprochen. Es wird empfohlen die Meßergebnisse in einer Strömungskarte zusammenzustellen. Eine Verlängerung oder Verkürzung der Kreislaufzeiten und Störungen der regionalen Durchblutung können erkannt und festgehalten werden. Über krankhafte Störungen des Hirn-Kreislaufes bei Gefäßerkrankungen und Geschwulsten wird berichtet. Abschließend wird auf die Möglichkeiten der Röntgen-Densitometrie zur Erforschung noch unbekannter Phänomene der Physiologie und Pathophysiologie des Hirn-Kreislaufes hingewiesen.

## SUMMARY

A new method for roentgen cinedensitometric analysis of the cerebral circulation is discussed. The preliminary results on the rate of flow, blood volume (in the internal carotid artery) and circulation time are reported. It is suggested that the various measurements might be compounded in a flow card; this would indicate any slowing down or acceleration of the circulation time as well as disturbances in the blood flow. The report covers vascular and malignant conditions. The possibilities of the method in research in certain physiologic or pathologic conditions are pointed out.

## RÉSUMÉ

Les auteurs exposent le principe d'une nouvelle méthode de roentgen cinedensitometrie pour l'analyse de la circulation cerebrale chez l'homme. Ils presentent les premiers resultats de leur recherche sur la vitesse de circulation sanguine, sur le volume du courant sanguin dans l'artere carotide interne et sur les temps de circulation du cerveau. Ils conseillent de rassembler les resultats des mesures en une carte de courants. Il est possible de reconnaître et de determiner l'allongement ou le raccourcissement des temps de circulation et les perturbations de la perfusion sanguine regionale. Les auteurs exposent leurs resultats sur les perturbations pathologiques de la circulation cerebrale dans les affections vasculaires et dans les tumeurs. Enfin les auteurs indiquent les possibilités d'utiliser la roentgen densitometrie pour l'etude approfondie de phenomenes encore inconnus de la physiologie et de la patho-physiologie de la circulation cerebrale.

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## MOTILITY OF THE CERVICAL VERTEBRAE IN LATERAL FLEXION OF THE HEAD AND NECK

by

J JIROUT

The mechanism of the vertebral reaction to lateroflexion of the head and neck in the sitting position was described by the author in 1967 and 1968 following earlier dynamic investigation. The skull rotates to the side of inclination with the vertex moving to this side and the base and occipital condyles to the opposite side. The axis of this rotation is located approximately at the level of the anterior cranial fossa (Fig. 1 a). The atlas is thus displaced to the side of the inclination although the opposite movement may occasionally occur.

The peculiar tilt of the articular facets at the level of C2—C3 is probably the underlying cause of the fact that rotation of the axis towards the side of lateroflexion commences immediately at the beginning of the lateral inclination of the head. During the continued movement this rotation increases possibly due to the unilateral pull of the cervicocranial muscles (rectus capitis posterior major and obliquus capitis inferior and superior) on the opposite side the tone of which is increased by sudden stretching. This pull acts on the spinous process of the axis and causes its rotation to the side of lateroflexion. The rotation of the axis is transmitted to the vertebrae below, this influence gradually decreases with the increasing distance from the axis but may sometimes reach as far distally as the upper thoracic region. The rotation is transmitted to the other vertebrae through

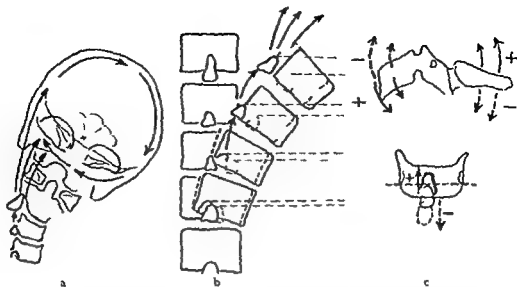


Fig. 1 a) Directions of forces (arrows) acting on the skull and cervical spine during lateral flexion of the head and neck (latent sitting). b) Direction of cranial pull on the spinous processes (arrows) and the resulting ventral tilt of the vertebrae with changes in projection of the spinous processes on the vertebral bodies during lateral flexion of the head and neck. + Ventral - and dorsal tilt of vertebrae. Lateral (upper) and a p (lower) views.

the normally shaped spinous processes and spinal ligaments. Congenital or acquired asymmetry of one of the spinous processes may prevent the rotary influence of the axis being transmitted in the lower parts of the spine.

The spinous process moves away from the side of lateroflexion and usually upwards; the latter movement results in the vertebra being tilted ventrally on a transverse axis through the intervertebral articulations (Fig. 1 b, c). Due to the relatively small size of the movements the possibility that they are artefacts caused by projectional distortion should be considered.

These dynamic phenomena were investigated in 326 patients under 45 years of age with various conditions but without obvious signs of spondylosis. The measurements were made in lateral views in the upright position as well as in lateroflexion in 104 patients and in a p views in 222 patients. The head and back of the sitting patients were in close contact with a supporting wall so that no sagittal movement of the upper part of the body was possible.

The degree of tilt of the tangent to the posterior aspect of the vertebral body or to the upper margin of the spinous process was determined in the lateral view (Figs 1c, 2, 3, 4). The projection of the apophysis of the spinous process and its relations to various structures including the lower margin of the vertebral



Fig. 2. Ventral tilt of C3 in lateroflexion of the cervical spine as measured by the angle between the tangent to the posterior aspect of the vertebral body and the vertical: a) Upright and b) lateroflexion position.

body was determined from the a.p. views. A tangent to the upper contours of the pedicles was usually used as the reference line (Figs 1 c, 6 to 8).

The changes in positions of 260 vertebrae were measured in the lateral view. A ventral tilt was evident in 214 instances, a dorsal tilt in 38 and no change in 28 instances. Tilting of the body and spinous process of the vertebra in opposite directions sometimes occurred; this probably pointed to the simultaneous influences of forces counteracting on different parts of a vertebra during lateroflexion of the spine.

Tilting of 2675 vertebrae during lateroflexion of the head and neck was measured in all the a.p. views. A ventral tilt was evident in 1278 vertebrae, a dorsal tilt in 497 instances and in 900 vertebrae no change occurred. Thus in nearly one half of the vertebrae a ventral tilt was observed while in the other half dorsal tilt and no tilt was recorded in the ratio of 1:2.

The possibility of the movements being artefacts due to differences in position or projection of the spine is improbable. Considerable differences in the degree of tilting of two neighbouring vertebrae in opposite directions could frequently be observed. Furthermore the tilt between two neighbouring segments that sometimes occurred strongly suggested the true nature of the movements (Fig. 8).

The influence of changes in position of the thoracic inlet on the upper thoracic and lower cervical vertebrae was examined in 252 measurements in 240 patients. It appeared that ventral or dorsal tilting of the thorax induces a corresponding displacement of the upper thoracic vertebrae only if the differences in projection



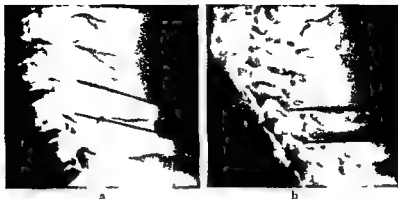


Fig. 3 Ventral tilt of C6 and C7 in lateroflexion of the spine demonstrated by changes in position of the tangent to the upper contour of the spinous processes: a) Upright and b) lateroflexion position



Fig. 4 Dorsal tilt of C7: a) Upright and b) lateroflexion position

of the clavicle on the posterior parts of the upper ribs were more than 5 mm. The cervical vertebrae do not appear to be influenced by changes in the position of the thorax which occur in association with lateroflexion of the cervical spine (Figs 6-7).

The dynamic investigations seem to indicate that lateral flexion of the head and neck tends to produce sagittal movements of the cervical vertebrae, in addition to their lateral inclination and rotation. This tendency is usually manifest by ventral tilting or sometimes by no tilt or even a dorsal tilt of the vertebrae. The extent of the movements of the vertebrae in the sagittal plane as evident



Fig 5 Ventral tilt of the axis in lateroflexion of the cervical spine as demonstrated by increased distance of the upper contour of the spinous process from the reference line connecting the lateral margins of the upper articular facets of axis. a) Upright and b) lateroflexion position

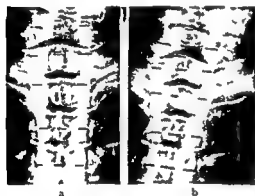


Fig 6 Ventral tilting of C—Th<sup>7</sup> vertebrae indicated by increase of the distance between the upper margins of the spinous processes and the reference line connecting the upper contours of the pedicles the position of the thoracic inlet is identical a) Upright and b) lateroflexion position

in the lateral views in the 104 patients was as follows. In 70 per cent of the 214 ventrally tilted vertebrae the tilt amounted to 5° or more in 58 it was 8 to 13 and in 8 vertebrae it was 14 to 18. In the 38 dorsally tilted vertebrae the change in position amounted in 50 per cent to 5 or more only in 7 instances did it reach 8 to 11. Further analysis indicated that the degree of ventral tilt of the C2—C3 vertebrae was greater than that of the C6—Th1 vertebrae. The percentage of vertebrae with a ventral tilt of 5 to 18 was 75 per cent at C2, 72 per cent at C3 while at C4—C7 it amounted to only 30 to 50 per cent. The apophysis of the spinous process was displaced upwards by 0.5 to 4 mm in most of the 1278 vertebrae with a ventral tilt. The greatest displacement (8 mm) occurred in 2 patients. If only the vertebrae with ventral or dorsal displacement of the spinous processes by 5 mm or more were considered it appeared that the greatest mobility lay at C4—C7.

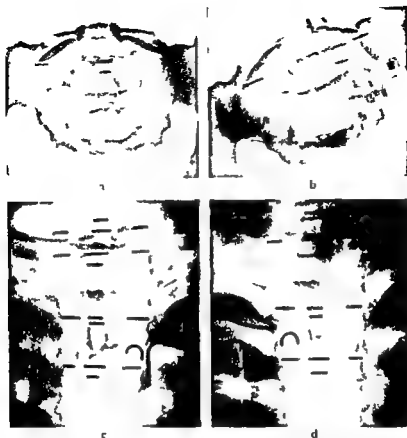


Fig. 1. Dorsal tilting of the axis: a) Upright and b) lateroflexion position. Ventral tilting of C7—Th3 vertebrae: c) Upright and d) lateroflexion position. The projection of the larynx on the posterior parts of the upper ribs is the same in both positions.

The importance of the absolute values is not to be overestimated. It would appear that the mutual relations of the variously directed components of tilt, namely ventral, dorsal or zero tilt, appear to be far more conclusive.

Ventral tilt appears to be much more frequent than dorsal tilt at C2 to C6. Both components, the ventral and the dorsal tilt, are equally frequent at the level of C7—Th1. The relative frequency of the ventrally tilted vertebrae decreases rapidly from C5 caudally, while that of the dorsally tilted vertebrae increases downwards. A mathematical statistical analysis of the results by means of the  $\chi^2$  test in which the degree of the tilt was compared in both directions at C2—C6 with that at C7—Th3 indicated that the differences between the ventral and dorsal tilting at various levels were highly significant.

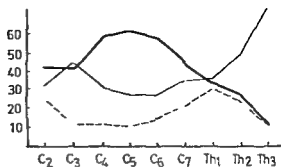


Fig 8 Dorsal tilting of C4 vertebra  
no tilting of C5 ventral tilting of C6  
in lateroflexion compared to normal  
a position

(Fig 9) It would thus appear that the frequency of dorsal tilt in the upper cervical segments down to C6 is much lower as compared with the ventral tilt and that from this level caudally its absolute and relative frequency increases. It is evident that this phenomenon is not fortuitous. It was therefore of interest to assess the dynamic components ventral + dorsal — zero 0, in the cervical spines of the material. The distribution of these components usually followed a certain system and appeared in various logical patterns. The values of the ventral tilt in several segments often gradually decreased to a zero zone from which the vertebrae were then increasingly tilted dorsally. For instance in one patient the values were Th1 — 4 mm C7 — 3 mm C6 0 C5 + 2 mm C4 + 3 mm C2 + 6 mm.

This distribution, together with the highly significant percentage increase of the dorsal tilt in the lower cervical segments seem to indicate that lateroflexion of the head and neck produces two dynamic effects upon the spinous processes and their ligaments. These consist in the action of the cranial pull causing ventral tilt and that of the caudal pull resulting in dorsal tilt of the vertebrae. The responses of the vertebrae to the action of these forces vary according to which prevail. In some subjects the whole cervical spine is under the influence of the cranial component in others the influence of the dorsal component asserts itself. These two components often meet at various levels at which the counteracting forces remain in balance a zero zone thus appearing. The localisation of this zero zone depends on where the two forces meet. It may be at various level more caudally if the cranial component prevails more cranially if the caudal component is stronger. The zero zone may extend through the whole cervical spine or again may be entirely absent (Fig 10). All these instances in which a certain logical pattern of the response to lateroflexion occurs belong to the group of harmonic response. In others the reaction fails to follow a

Fig. 9 Percentage distribution of ventral (—), dorsal (---) and zero (—) components at various levels of cervical spine



logical pattern and their reaction may be referred to as a disharmonic response.

The vertebral tilts were measured in an adequate number of segments in a group of 416 patients so as to render the evaluation of the reactional pattern possible. A total of 722 lateroflexions were measured and in about three quarters a harmonic response was observed.

### Conclusions

Certain complex dynamic phenomena occur during lateroflexion of the head and neck. The cervical vertebrae are subject to counteracting forces the action of which results in inclination, rotation and sagittal tilting of the vertebrae. The active pull of the cervicocranial muscles on the spinous process of the axis is

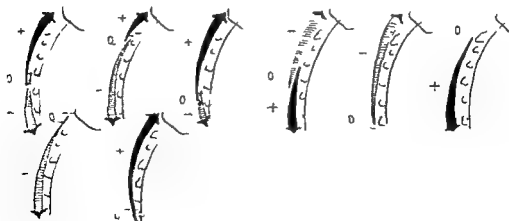


Fig. 10 Various patterns of the harmonic response of the cervical vertebrae on lateroflexion of the head and neck.

transmitted downwards and seems to prevail over the more passive opposition of the caudal pull that results from ligamentous resistance. If these components stay in balance, no tilt results. However, even if no change is apparent, the action of these forces still remains. A definite logical pattern of vertebral response to the lateroflexion may usually be observed.

As no further physiologic movement of the vertebrae can occur in maximum lateroflexion with associated passive rotation and considering the various structures opposed to such additional movement, it is remarkable that the sagittal tilt of vertebrae can be demonstrated at all. It would appear that the forces capable of eliciting this dynamic component are by no means negligible and that they must be considered when any sort of manipulation is applied to the cervical spine.

## SUMMARY

The examination of 2905 vertebrae in 326 patients indicated that ventral or dorsal sagittal tilting of the cervical vertebrae occurs during lateral flexion of the head and neck. The complex dynamic phenomena are discussed.

## ZUSAMMENFASSUNG

Die Untersuchung von 2905 Wirbeln von 326 Patienten weist darauf hin, dass eine ventrale oder dorsale sagittale Neigung der Halswirbel während der lateralen Beugung des Kopfes und Nackens erfolgt. Dieses komplexe dynamische Phänomen wird besprochen.

## RÉSUMÉ

L'examen de 2905 vertèbres chez 326 sujets a montré que les vertèbres cervicales s'inclinent en avant ou en arrière au cours de la flexion latérale de la tête et du cou. L'auteur étudie ces phénomènes dynamiques complexes.

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## CEREBELLOPONTINE CISTERNOGRAPHY WITH A POSITIVE CONTRAST MEDIUM

by

JACK LESTER

New surgical approaches in the radical treatment of acoustic neuromas have made early diagnosis with establishment of the exact size of the tumour necessary. Good results have been obtained in air cisternography with a Polytome (Stew et coll 1968) but as it is difficult to recognize the small tissue structures in the cerebellopontine cistern or internal auditory canal the present author prefers a positive contrast medium. A method of cisternography with Pantopaque based on experiences from the Otologic Medical Group of Los Angeles (HITZBERGER & HOUSE 1968; BLITTON et coll 1968) has been developed during the last two years. The preliminary results of this method are now presented.

*Technique.* The cerebellopontine angle and the internal auditory canal are filled with 1.5 ml Pantopaque by tilting the patient 15° for five minutes in the lateral decubitus position on a slightly modified Fleury-Schönander easurely table without fluoroscopy. Films are obtained with a CRT 7 skull unit in the following projections: (1) Stenvers, with the patient prone and the head rotated 45°; (2) half axial (Fig. 1); (3) transorbital and (4) submentovertical. The last three films are taken with a horizontal roentgen beam with grid craters and the patient lateral.



Fig. 1 Half axial view of normal case Pantopaque in the cerebellopontine cistern and internal auditory canal

Incomplete filling of the auditory canal indicates supplementary tomographic views in Stenvers or better still in the tran orbital projection where the canal is projected in its maximum length although are not part of the routine examination. These cuts are easily produced with the CRT 7 skull unit by either linear or circular movements. It may also be necessary with questionable filling defects to examine the opposite normal side. This is done after transferring the Pantopaque to the other cerebellopontine cistern by extension and rotation of the head. When the examination is concluded the Pantopaque will run back into the spinal canal when the patient sits up if the chin be hyperextended for a few minutes no attempt is made however to remove the small quantity as it may if necessary be used again for control.

*Material* Fifty cisternographies with Pantopaque were performed over two years in 21 men and 29 women the youngest being a 13 year-old girl with Recklinghausen's disease and bilateral acoustic neuromas and the oldest a 75 year old woman about 60 per cent of the patients were aged between 40 and 60.

### Results and Discussion

The 50 examinations revealed 17 tumours in 13 women and 4 men. Three refused operation but in the other 14 patients 11 acoustic neuromas and 3 meningiomas were removed. Encephalography was performed in 5 patients with positive cisternography but although a pathologic process was diagnosed in all 5 it was not possible to determine exactly the size of the tumour. Vertebral angiography was also carried out in 4 patients with a positive result but failed to furnish exact information as to tumour size. Dividing the 11 acoustic tumours into 3 groups (HITZELBERGER & HOLME 1966) (1) small (confined to the canal), (2) medium (extending out of the porus but only with eighth nerve dysfunction) and (3) large (with other cranial nerve findings) the series comprised 11 medium and 5 large, but no purely intracanalicular tumours.



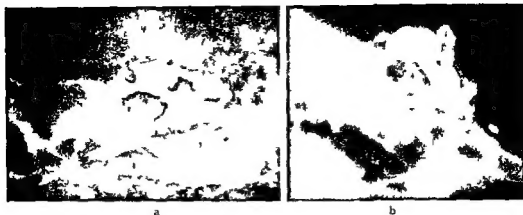


Fig 2 Medium sized tumour a) Stenvers and b) half axial views. No filling of the canal. Tumour mostly intracanalicular but slightly protruding into the cistern. Operation revealed acoustic neuroma about 1.0 cm in diameter.

The method has proved very useful in the diagnosis of the medium (Figs 2 and 3) as well as the large (Fig 4) tumours. In all the 11 patients with acoustic neuroma verified by operation it was preoperatively possible from the four different views to determine the exact size of the growth. Good correlation between its size in the film and at operation was evident in the 3 patients with meningiomas.

The small quantity of Pantopaque (1.5 ml) employed makes superimposition of the medium in the cistern no problem in evaluating the internal auditory canal. As the patient lies most of the time in the lateral decubitus position there is no danger of the contrast medium spilling into the middle cranial fossa. Good filling of the internal auditory canal occurred in all the 33 normal patients including one in whom irregular configuration of the contrast medium in the cerebellopontine cistern suggested leptomeningeal adhesions. The vertical position of the canal in the lateral position of the patient affords an optimal chance of its filling although penetration of the contrast medium to the meatal fundus cannot be expected in all normal instances (Fisch 1969). This is probably due as much to the presence of the vestibular ganglion as to wide variation in the size of the intracanalicular subarachnoidal space (VALVASSORI 1969). The column of contrast medium in normal instances tapers laterally in the canal on both sides of the falciform crest (Fig 1) but a small intrameatal neuroma will produce a smooth indentation. Doubtful filling defects must therefore be further investigated by tomography.

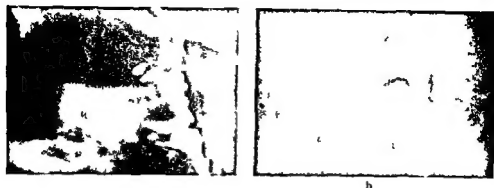


Fig 3 Medium sized tumour clearly outlined in a) transorbital and b) submentovertical views. Operation revealed acoustic neuroma 1.5 cm in diameter.



Fig 4 Stenvers view of large cerebellar pontine tumour. Operation revealed acoustic neuroma, 4 cm in diameter.

### Conclusion

Cisternography with Pantopaque as contrast medium has in 50 patients proved most valuable in the diagnosis of medium as well as larger tumours. The technique with only 15 ml of Pantopaque and without fluoroscopic control should reveal the nature and determine the exact size of all lesions in the cerebellopontine angle.

### SUMMARY

The technique and results of 50 positive contrast cerebellopontine cisternographies made with 15 ml Pantopaque on a skull table without fluoroscopy are described. Four different views were used but tomography was not a part of the routine.

## ZUSAMMENFASSUNG

Die Technik und die Ergebnisse von 50 positiven cerebello pontinen Kontrast Cisternographien mit 15 ml Pantopaque auf einem Schädeltisch ohne Durchleuchtung ausgeführt werden beschrieben. Vier verschiedene Darstellungen wurden verwendet. Aber die Tomographie wurde nicht routinemässig vorgenommen.

## RÉSUMÉ

Description de la technique et des résultats de 50 cisternographies ponto cerebelleuses a contraste positif faites avec 15 ml de Pantopaque sur un craniographe sans radioscopie. L'auteur utilise quatre incidences différentes. La tomographie n'est pas utilisée de façon systématique.

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